THE JOURNAL OF PERIODONTOLOGY

January
1953

Published by
THE AMERICAN ACADEMY OF PERIODONTOLOGY
VOLUME 24
NUMBER 1

RK 1
J855
v.24

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THE JOURNAL OF PERIODONTOLOGY is published to serve as the professional journal of periodontia; to keep dentists in touch with research being carried on in this field and related sciences; to stimulate greater interest in the supporting tissues of the teeth and their relation to oral and general health.

The Journal is the official organ of the American Academy of Periodontology and will contain, therefore, papers of scientific and clinical interest which are presented at the annual meetings of that society, as well as articles submitted direct to the Editor. Obviously opinions expressed do not necessarily represent those of the Editor or the American Academy of Periodontology.

Thirty-seventh Annual Meeting
THE AMERICAN ACADEMY OF PERIODONTOLOGY
September 24, 25 and 26, 1953
Cleveland, Ohio

Annual Spring Seminar
April 27, 28, 29, 1953
Ann Arbor, Michigan
See page 59 for details

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THE JOURNAL OF PERIODONTOLOGY

JANUARY, NINETEEN HUNDRED FIFTY-THREE

Systemic Aspects of Periodontal Disease

by LESTER W. BURKET, D.D.S., M.D.,* *Philadelphia, Pa.*

IT IS generally recognized that multiple factors are etiologic components for most cases of periodontal disease and that these may include systemic as well as local causes. While there must be some systemic participation in all patients with periodontal disease, clinically it is much easier to demonstrate the etiologic significance of local factors in the causation of the periodontal lesions and in response to treatment. It is entirely illogical to assume that systemic factors are important only in patients with periodontosis. The problem for consideration is the relative importance of systemic as compared with local factors. It is difficult to recognize these systemic factors by means of clinical symptoms or current laboratory tests and it is difficult to evaluate the relative clinical importance of systemic as compared to local etiologic factors, or the relative significance of any particular systemic disease.

This subject was discussed in detail by numerous authorities at the 1951 Periodontal Workshop held at Ann Arbor, Michigan. In the editorial comment concerning this Workshop it was stated that "A critical evaluation of the available reports shows that the significance of specific systemic factors as etiologic components in the pathogenesis of marginal periodontitis has not been established."[1] One year later this statement can not be further qualified.

There is probably no branch of dental practice that requires a more thorough evaluation of the general health of the patient than periodontology. The health status of the patient is essential not only to determine the etiologic factors, to plan the treatment but also to establish the prognosis. Because of their unique anatomic relations and local environment, the periodontal tissues may be the first in the body to reflect changes in the over-all health status of the patient.

The evaluation of the possible role of systemic factors in periodontal disease has been approached experimentally by means of studies on animals. In many instances the severity of the experimentally produced systemic disease was not comparable with that found in man, or the rarity of the condition minimized general clinical significance of the observations. Even in those studies in which definite changes were noted in the periodontal tissues, it can not be assumed that the naturally occurring derangements in man give rise to similar changes. These studies however, furnish important leads for careful and extensive clinical obser-

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vations and investigations.

Attempts have been made in the clinic to evaluate the importance of systemic factors in periodontal disease. These have included blood and urine chemistry analyses and other common hematologic studies in groups of patients who have periodontal disease as compared with patients without periodontal disease.[2,3,4] Tests evaluating the activity of the various endocrine glands have also been employed.[5,6] The results of physical examinations or medical case histories in these two groups of individuals have also been analyzed. In some specific tests considerable variation is observed in the individuals with periodontal disease, but the abnormal findings are so scattered and unrelated to one another that it is obvious on inspection that there is no one systemic disturbance that is common to the patients with periodontal disease. This statement should not be interpreted to imply that systemic disease is not important in the periodontal patient; in fact the opposite. It indicates the need for a more thorough physical evaluation of these patients.

For over two years, all patients being treated in any of the clinics of the School of Dentistry of the University of Pennsylvania have been required to fill out a Health Questionnaire which is devised to furnish a general idea of the health status of the patient, both past and present.[7] This technic has been found to be very useful as a case finding tool. Five hundred of these Health Questionnaires selected at random from patients with periodontal disease and a similar number from dentulous patients without periodontal disease, were tabulated to determine whether there was any pattern of answers which was characteristic for the patients with periodontal disease. The Health Questionnaires of the patients with periodontal disease presented no evidence that any particular systemic disease or group of diseases was associated with the periodontal disease.

The systemic conditions which are potential factors in periodontal disease can be grouped in several categories for purposes of discussion.

1. Nutritional disorders
2. Endocrine disorders
3. Diseases of the blood and blood forming tissues which may be associated with periodontal changes.
4. Other diseases which may be associated with periodontal changes, and those where treatment may produce periodontal changes.

NUTRITIONAL DISORDERS

Nutritional disorders, recognized or unrecognized, are without question the most common and hence perhaps the most important group of systemic diseases which may affect the periodontal tissues. We should be concerned with the more frequent mild, chronic nutritional disturbance rather than the severe acute deficiencies. It should also be stressed that in addition to adequate food intake, good nutrition requires proper absorption and utilization of the food.[7] These later phases of nutrition have been too frequently disregarded by the physician and dentist and in the age range when periodontal disease is most prevalent they are of great importance.

Experimental studies have clearly demonstrated the many ways nutritional deficiencies may affect the tooth supporting tissues. Many of the earlier experiments employed procedures which were extreme in degree and hence not comparable to those which might be encountered in the clinic, or they were confined to a study of rare nutritional disorders of little clinical significance.[8] It is unlikely for example that the severe experimental vitamin A deficiency states would be found in man in this country. It is also unlikely that the effects noted by Glickman on animals on a starvation diet would be encountered.[9] These extreme experimental studies however, do indicate what may occur to a lesser degree in a milder form of deficiency.

Some of the experimental studies have

been concerned with mild, chronic deficiencies such as one might expect to find in clinical experience. The mild, chronic C deficient animals studied by Boyle[10] presented both the gross and microscopic periodontal findings similar to those found in man. These lesions were produced in experimental animals who do not under natural conditions develop periodontal disease. All of these studies tend to emphasize the wide range of systemic conditions which may involve the periodontal tissues and the need for more studies, including good clinical observation and analysis. There is a considerable scientific gap between the animal room, the pathologist's laboratory and the clinic or the dental office and one must be cautious about making direct inference from these experimental studies.

Many clinical studies have been carried out in an attempt to evaluate the role of nutritional deficiencies in the development of periodontal disease. Food intake studies reveal no marked differences in periodontal patients from control individuals.[11] They do, however, stress that a considerable percentage of the population have a deficient food intake according to generally accepted standards. The results of the average diet study furnish at best only a rough estimate of one part of the nutritional status of the patient, the adequacy of the food intake.

Urinary determinations of thiamin, riboflavin and nicotinic acid made by Karshan[4] show no evidence of any subclinical nutritional deficiencies in the patients with periodontal disease. There is some evidence that plasma ascorbic acid levels are lower than normal in a large proportion of the patients with periodontal disease and that calcium intakes are lower.[12,13] These findings do not mean that prescribing additional amounts of vitamin C or calcium alone will have a beneficial affect on any periodontal disorder.

The early recognition of nutritional deficiencies is an important problem in medicine and dentistry. As yet no simple tests are available, which will permit the detection of mild deficiency state before the gross clinical lesions develop. Thirteen hundred patients receiving periodontal treatment at the School of Dentistry, University of Pennsylvania were carefully studied for any of the signs or symptoms suggestive of nutritional deficiency which were listed by the Food & Nutrition Board of the National Research Council. A control group of comparable number and age distribution were also studied. The only finding peculiar to the patients with periodontal disease was the greater prevalence of "bleeding gums." This finding could be readily explained by local irritative factors in the periodontal group. When the clinical criteria generally accepted as indicating nutritional disturbances were used, the patients with periodontal disease were not observed to have any increased incidence of nutritional disorders.

The preceding discussion should not be interpreted to infer that good nutrition is not important for the maintenance of healthy periodontal tissues. With the more general appreciation of the constant replacement and repair which are continually taking place in the tooth supporting tissues, the importance of good nutrition assumes greater significance. Moreover good nutrition is not something that can be achieved by prescribing a few vitamin capsules for several weeks. Nutritional problems, in the majority of adult and elderly patients, result from disturbances in absorption and utilization and hence are primarily the responsibility of the physician.

ENDOCRINE DISORDERS

Endocrine or hormonal disorders may be associated with changes in the periodontal tissues. Some of these result in marked damage to the tooth supporting tissues, however, because of their rare occurrence they are of little clinical importance. Hyperparathyroidism is an example of an endocrine disturbance which may be associated with both gingival and periodontal lesions.[14] Fortunately this disease is rare. Any patient presenting recurrent epuli, particularly when these growths involve

different areas of the gingival tissues should be suspected of having hyperparathyroidism. These soft tissue masses on the gingiva may appear as long as two years before the true nature of the disease is recognized.[15] The soft tissue growths should be examined histologically and roentgenograms should be taken to rule out osseous lesions.

Early loss of teeth is a common finding in hyperparathyroidism. This results from the demineralization of the alveolar process, loosening of the teeth and associated inflammatory changes in the gingiva and periodontal membrane. There does not appear to be any demineralization of the teeth. Multiple bone cysts in the jaws may also be found in this disease. The diagnosis of hyperparathyroidism is made on the basis of the typical blood chemistry findings and skeletal xray studies.

Disturbances in the sex hormones, especially in the female, are said to result in gingival changes.[16,17] There is little doubt that clinical and microscopic changes can be observed in the oral tissues in some postmenopausal women. Younger women who have marked ovarian dysfunction may present an inflammatory type of gingival enlargement and gingival tissues that are painful and easily traumatized. Since these tissues are difficult to maintain in a satisfactory state of oral hygiene and since they are susceptible to secondary infection they may predispose to more serious periodontal disease. These patients, however, represent a small percentage of all the patients with periodontal disease.

The gingival changes associated with pregnancy and at times menstruation are related in part to endocrine disturbances.[18,19,20] While rather striking soft tissue lesions may be observed in some pregnant women, they are comparatively rare in those who have good oral hygiene and periodontal care. As Kerr suggests, they probably represent a form of granuloma pyogenicum.[21] It does not appear that these disturbances are important systemic factors in predisposing to periodontal disease. Numerous clinical and laboratory studies, including our survey of 900 consecutive admissions to the periodontia clinic, have failed to substantiate any clinically significant relation between gonadal disturbances and periodontal disease.

The clinical effect of adrenal disturbances on the tooth supporting tissues have not been reported, probably because the life expectancy of patients with tumors of this endocrine gland are comparatively short and any periodontal lesion that might develop is overshadowed by the general symptoms. It has been shown experimentally, however, that the administration of cortisone to normal experimental animals (mice) has resulted in osteoporosis of the alveolar process, degeneration of the collagen fibres of the periodontal membrane, capillary enlargement and even hemorrhage into the periodontium and gingiva.[22] Clinical observations on the deleterious affect of ACTH or cortisone in the development or treatment of periodontal disease have not been reported.

UNCONTROLLED DIABETES

The endocrine disturbances associated with uncontrolled diabetes have long been considered to be an important systemic factor in the causation of periodontal disease. This association between periodontal disease and uncontrolled diabetes has been mentioned by physicians for many centuries. One of the good early clinical studies on this subject was made by Aiguier.[23] Rapidly developing periodontal disease may be the clinical findings which first cause one to suspect uncontrolled diabetes, this is especially true in the adolescent. There is a real question whether there are any characteristic features of the gingival or periodontal lesions in the uncontrolled diabetic which permit the diagnosis of this disease on the basis of the oral findings. The fungating masses of granulomatous tissue which protrude from the periodontal pocket, as described by Hirschfeld, are not commonly found.[24] The periodontitis may

have a tendency to be more painful than that which is usually observed and to be associated with suppuration.

The relationship between the periodontal, gingival and oral mucosal lesions frequently observed in the uncontrolled diabetic patient may be indirect rather than direct. The effect of the diabetic state on the periodontal tissues has recently been investigated experimentally by Glickman in alloxan induced diabetes.[25] No changes were observed in the gingival tissues or the periodontal membrane of the experimental animals, although there was definite evidence of demineralization of the alveolar process.

The possibility of an indirect effect of the uncontrolled diabetic state on the development of periodontal disease has been mentioned previously. It is recognized that these patients have a lessened ability to combat infection, hence, any role that nonspecific bacterial factors might play in the development of periodontal disease would be accentuated. This might explain in part the common painful and suppurative nature of periodontal disease in uncontrolled diabetes.

Another indirect and perhaps important factor in the development of periodontal disease in the uncontrolled diabetic is the depressed activity of vitamin C and the increased requirement of vitamin B complex, which is associated with the diabetic state; both conditions which may have a detrimental affect on the tooth-supporting tissues. When the periodontal, oral mucosal and lingual findings associated with uncontrolled diabetes are compared with those occurring in vitamin B complex deficiency, the similarity of the symptoms is obvious. Rudy and Hoffman[26] believe that the dermatologic symptoms of diabetes are adequately explained on the basis of a conditioned nutritional deficiency. This may also hold true for the oral lesions. There are other factors in the uncontrolled diabetic state which may predispose to the development of periodontal disease. The reduced alkaline reserve in the diabetic patient may favor the deposition of calculus with its local irritant effect on the periodontal tissues.

While the specific and direct effect on the tooth supporting tissues of the uncontrolled diabetic state may be limited to a demineralization of the alveolar process, as shown experimentally by Glickman, indirect effects in man which are related to rapid and abundant calculus deposits, decreased resistance of the oral tissues to nonspecific bacterial invasion and conditioned nutritional deficiencies appear to be of clinical importance. Unrecognized diabetes should always be ruled out in the patient with periodontal disease, especially a rapidly developing form of the disease in adults and when it is found in the adolescent, Rutledge[27] has shown that in children, where severe periodontal disturbances are not common, that uncontrolled diabetes is an important predisposing factor. The testing of the urine for reducing substances is one laboratory procedure which should be routine for the periodontal patient.

Disturbances of the thyroid gland, both hyper and hypothyroidism have been said to predispose to periodontal disease. Alveolar absorption has been observed in experimentally produced hyperthyroidism in guinea pigs. Increased vascularity of the alveolodental periosteum was noted and increased osteoclastic activity along the compact layer of the bone surrounding the tooth. Again, as in hyperparathyroidism, usually the general systemic symptoms would result in early recognition of the hyperthyroidism, with treatment of this condition before marked destructive changes have developed in the periodontal tissues. The systemic symptoms in hypothyroidism are less striking and it is possible that mild hypothyroid states may exist for a considerable period of time before they are recognized.

Diseases of the Blood and Blood Forming Tissues Which May Be Associated with Gingival and Periodontal Changes.

Many of the diseases of the blood and

blood forming tissues affect the gingiva and periodontal structures.[28] The first symptom of malignant neutropenia may consist of ulcero-necrotic lesions of the cheek, pharynx or the marginal gingiva. These moderately painful ulcerations frequently have a greenish-black base. These patients also complain of a "woody sensation" to teeth and rapidly developing looseness of the teeth. A definite diagnosis of malignant neutropenia can not be made on the basis of the clinical findings, since hematologic studies are always required. The importance of early diagnosis in this disease cannot be overemphasized to the dentist since the earlier therapy is instituted the more favorable is the prognosis.

The first clinical symptoms of the acute leukemias, particularly acute monocytic and myelogenous leukemia, may be noted in the mouth. They consist of a marked enlargement and frequently necrosis of the marginal gingival tissues, the cheek and at times the tongue. These patients commonly first consult their periodontist in the belief that the gingival changes are of local causation. The earliest lesions are usually found in areas where there is some form of chronic irritation. The maxillary molar region and the mandibular anterior region are common locations.

The marked gingival enlargement is caused by the edema and necrosis of the tissues and the marked extravascular collections of immature, abnormal leukocytes.[29] It is interesting that the oral lesions are only found in those forms of leukemia in which the mature cell type occurs extravascularly. Small thrombi may form in the vessels supplying the gingival tissues and predispose to the development of large necrotic areas. Since these immature leukocytes do not have the same ability to combat infection as mature cells, these areas of gingival enlargement are particularly susceptible to secondary infection with the oral flora.

All the other soft dental tissues may be similarly involved in the leukemias. The extra-vascular accumulation of these immature leukocytes in the periodontal membrane, result in rapid loosening of the teeth. Thrombi can be observed in the vessels in the periodontal membrane and differentiation of the collagenous fibres becomes less distinct as these structures are destroyed. The pulpal tissue is also involved in the same process. Patients with monocytic and myelogenous leukemia may experience pain and/or spontaneous pulpal hemorrhage in clinically sound teeth. Liquefaction necrosis of the pulp may result in periapical abscess and fistula formation.

It is important that the clinician recognize the systemic basis for these symptoms so that instrumentation or surgical procedures will not be undertaken in these patients. In the leukemias, as with malignant neutropenia, the correct diagnosis can only be established by hematologic studies and frequently bone marrow smears. Since at present there is no effective treatment for the leukemias, the periodontist's role in the handling of these patients consists in keeping the patient's mouth comfortable and reasonably clean. The usual dental prophylaxis and periodontal treatment or oral surgical procedures are contraindicated. Simple non-irritating mouth washes such as sodium bicarbonate are useful for maintaining the oral hygiene. Antibiotic therapy, both topically and by the parenteral route greatly lessen the tendency for the painful, foul smelling necrotic lesions to develop.

Polycythemia vera may also be associated with an enlargement of the gingival tissues which have a bluish-purple, cyanotic color. In this condition there may be persistent bleeding from the gingival tissues and particularly after the extraction of teeth. The gingival lesions are important only as a means of recognizing the underlying systemic disease. With modern methods of therapy the life prognosis in this disease is reasonably good.

Every dentist is aware of the oral aspects of hemophilia. Many individuals with this disease are first aware of its existence as

the result of complications following the eruption of teeth or following oral surgical procedures. Periodontal lesions are not a prominent part of the oral symptomatology, and this has been difficult to explain, since spontaneous hemorrhages into the joints is one of the characteristic and crippling features of this disease. The blood vessels in the periodontal membrane must be better protected from trauma than those in the joints. Hereditary familial telangiectasis is a rare systemic disease which may be associated with telangiectatic areas on the gingiva and oral mucosa. These have a tendency to copious bleeding, at times without any recognized provocation.

Other Diseases Which May Be Associated With Periodontal Changes, and Those Whose Treatment May Produce Periodontal Changes.

There are numerous systemic diseases that may predispose to periodontal lesions, or whose treatment may be associated with periodontal disease. The therapeutic administration or occupational exposure to any of the heavy metals may produce gingival and periodontal disorders. Mercury, bismuth and lead are the heavy metals most likely to be associated with periodontal changes. Some of the salts of these metals which are eliminated in the saliva, particularly mercury, may be irritating to the oral mucosa. Their major effect on the gingival and periodontal tissues is, however, an indirect one. The sulfide salts of these metals become deposited in the marginal gingiva and interdental papillae in areas where the oral hygiene is poor and there is bacterial decomposition of food remains. The salts which are deposited in the capillaries of the marginal gingiva and the surrounding tissues interfere with the nutrition of the tissues, rendering the gingiva more susceptible to secondary infection by the bacterial flora of the mouth.

A decade ago bismuth gingivo-stomatitis was common observed in patients receiving the usual antiluetic treatment. Not only did the ulceronecrotic gingivostomatitis which developed cause potential and real injury to the periodontal tissues but at times it necessitated the discontinuance of the bismuth injections. Today with the more general use of antibiotics for the treatment of syphilis, bismuth gingivostomatitis is of less clinical importance.

The most severe injury to the periodontal tissues may be found in association with the administration of mercurial salts or occupational exposure to the vapors of this metal. Mercury inunctions, another old treatment for syphilis, are rarely used in modern medicine. While the occupational hazards of mercury vapor have been markedly eliminated, new sources of exposure are continually appearing. A recently recognized mercurial hazard appeared in the fluorescent lamp manufacturing industry. It is interesting that this industrial hazard was recognized by the development of a painful marginal gingivitis and periodontitis among the workers. The oral tissues in patients with mercurialism are particularly susceptible to secondary infection and when traumatized extensive sloughing areas may develop which can involve the alveolar process as well as the gingival and periodontal tissues.

Plumbism is almost entirely an occupationally acquired condition. While the lead line is of great diagnostic significance, the systemic symptoms of lead intoxication usually overshadow those related to the mouth.

Dilantin sodium is extensively used in the treatment of the grand mal type of epileptic seizure. One of the important and unexplained side effects of this drug is the gingival enlargement which develops in varying degrees and frequency in these patients.[30] This fibrotic type of gingival enlargement can not be directly correlated with the dosage or the duration of administration of the drug although it is rare to find this gingival change developing until it has been administered for 2 or 3 months. It does not appear to be related to the vitamin C nutrition of the individual. Good, conscientiously carried out oral hygiene from the time the dilantin is first pre-

scribed appears to be the best preventive.

This type of gingival hyperplasia is of great interest to the periodontist and the physician. In its fully developed state, surgical removal is usually indicated and this by no means assures against the regrowth of the excess tissue.[31] As in so many diseases, prophylaxis is the best method of treatment. Attempts are now being made to study the mechanism of this stimulation to connective excessive tissue growth by dilantin through tissue culture studies, in the hope that more effective preventive measures can be taken and that the therapy of dilantin hyperplasia will be more successful.

Another disease which appears to affect only the gingival tissues and one which may exhibit hereditary tendencies should also be mentioned, namely; diffuse fibromatosis of the gingiva.[32, 33] The exact cause of this condition is not known. It may occur sporadically or be present as an inherited characteristic. While it occasionally appears during the period of the deciduous dentition, it usually becomes clinically recognizable during the eruption of the permanent dentition. Many times the teeth never appear to erupt completely through the hyperplastic gingival tissues. Aside from the unesthetic appearance, and the difficulty in chewing which arises in these patients, few if any symptoms are experienced. In both dilantin hyperplasia and diffuse fibromatosis there may be little or no involvement of the interseptal bone during a period of several years in contrast to the early involvement of the interseptal bone in the inflammatory type of gingival enlargement. Simple removal of the enlarged tissue by gingivectomy does not usually produce a permanent result. A combination of surgery and radiation therapy as recommended by Ball[34] may be effective.

SUMMARY

Since the periodontal structures are part and parcel of the entire organism they must participate to some degree in every systemic derangement. Our present inability to find one or several systemic conditions which may serve as etiologic factors in periodontal disease should emphasize the need for complete physical evaluation of every patient.

The chronic mild nutritional deficiencies represent probably the most important group of diseases which may predispose to periodontal disorders. Uncontrolled diabetes has an indirect and perhaps a direct relation to periodontal disturbances. Every periodontist should serve as a "case finder" for this increasingly common disease.

Disturbances of endocrine function and some of the diseases of the blood have a marked effect on the tooth supporting tissues, however, their rare occurrence minimizes their general clinical significance. The side effects of therapy of many systemic diseases and the metal intoxications result in distinctive periodontal lesions.

The exact significance of specific systemic factors as etiologic components in the pathogenesis of periodontal disease can not be determined. This, however, does not justify a disregard for systemic factors in patients with periodontal disease.

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DEATH OF DR. GRACE ROGERS SPALDING ANNOUNCED

We are sorry to inform our readers that Dr. Grace Rogers Spalding, first Editor of the JOURNAL OF PERIODONTOLOGY, died January 7, 1953. Since this information was received just as the Journal was going to press, details cannot be included in this issue but will appear in the April issue.

The Attachment of Calculus to Root Surfaces*

by HELMUT A. ZANDER, D.D.S., M.S., *Minneapolis, Minnesota***

CALCULUS consists of an organic matrix and mineral precipitates. The organic matrix is formed by microorganisms, of which the actinomyces are the predominant forms.[1] Several theories have been advanced regarding the mode of precipitation of the minerals from saliva and their deposition into the organic matrix.[2]

An important aspect of the problem of dental calculus is its attachment to the tooth surface. Little is known about calculus attachment beyond the statement that bacteria play an important role in the attachment of calculus. It is a well established clinical observation that the removal of calculus from the root surfaces of teeth is sometimes very difficult and at other times rather easy. This difficulty or ease of calculus removal might be dependent upon the mode of attachment. Since detachment of calculus is one of the prime objectives of periodontal treatment, the mode of attachment of this structure needs investigation; therefore, this study was undertaken.

PROCEDURES

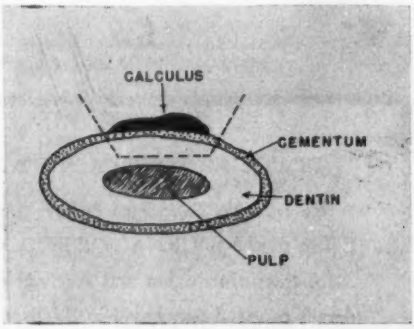
Immediately after extraction of a tooth with gross calculus on its root surfaces, two cuts were made with a double sided diamond disc on either side of the calculus, and a piece of the tooth was cut out (Figure 1). Thus a specimen was obtained consisting of dentin, cementum and calculus. This procedure was carried out on 50 teeth. Some of the specimens were fixed in Zenker-Formol solution and decalcified in 10% formic acid over a small amount of an ion exchange resin.[3] An alternate method used combined fixation and decalcification using formalin and trichloroacetic acid.[4] The specimens were embedded in celloidin, sectioned and stained with hematoxylin and eosin, and with a bacterial tissue stain.[5]

RESULTS

Four modes of attachment of the organic calculus matrix to the root surface were observed. One: Attachment by means of the secondary cuticle (Figure 2). The dental cuticle is believed to be formed by the epithelial attachment as it contacts the cementum. It remains attached to the cementum when this epithelium separates from the root surface. It is then exposed to the saliva and the microorganisms which make up the calculus matrix. Apparently these microorganisms can attach themselves to the cuticle.

Two: Attachment to the microscopic irregularities of the cementum surface (Figure 3). The dental cuticle is often destroyed by microorganisms in the mouth or by the dentist in giving an oral prophylaxis. The calculus matrix is then attached to the minute spaces of the cementum which correspond to the previous location of Sharpey's fibers.

Fig. 1. Method of obtaining specimen.



*Presented before the annual meeting of the American Academy of Periodontology, St. Louis, Missouri, September, 1952.

**From the Department of Periodontology, University of Minnesota School of Dentistry, Minneapolis 14, Minnesota.

Fig. 2. Calculus attachment to secondary cuticle. (900×) A—Cementum B—Cuticle C—Calculus

Fig. 3. Calculus attachment to the irregularities of the cementum surface. (1000×) A—Cementum B—Calculus

Three: Attachment by penetration of microorganisms making up the calculus matrix into the cementum. (Figure 4). These microorganisms may be found in the outer part of the cementum and may also have penetrated to various depths.

Four: Attachment into areas of cementum resorption. (Figure 5). Resorption of the cementum often occurs with or without various degrees of repair. In periodontal disease these areas become exposed to the saliva by pocket formation. They often present undercuts, and when calculus forms here a mechanical locking occurs.

There is seldom a uniform mode of attachment of one piece of calculus. A combination of some of the four modes described commonly exists. Table 1 gives the distribution of the types of attachment for the 50 teeth studied. It may be seen that combinations which include modes III and/or IV are the most frequent.

DISCUSSION

Obviously the location and the hardness of calculus have something to do with the ease or difficulty of its removal. However, it is only logical that on the detachment of calculus, a very important factor is its mode of attachment. It is readily understandable that if the calculus is locked in an undercut resorption area of the cementum (Figure 5), its removal would be extremely difficult or even impossible without removal of some of the cementum. Dislodgment with a scaler would probably result in breaking off the calculus from the outside of the cementum leaving the anchored part. This would make reattachment of the soft tissue impossible and calculus will again develop and continue to be an irritant to the adjacent gingival tissues. Therefore curettage, as it is understood by the periodontist, with its implication of cementum removal is a "must" in this case. On the other hand, if the

TABLE I

The Incidence of Mode of Calculus Attachment to Cementum

I Via secondary cuticle	*II Direct cementum surface attachment*	*III Penetration into cementum*	*IV Mechanical retention in areas of resorption*	*V (Combination of III & IV)*	*VI (Combination of II, III, IV)*	*VII (Combination of I, II, III, IV)*	*VIII (Combination of I & II)*	*IX (Combination of II & III)*	*X (Combination of II & IV)*
0	10	5	0	6	7	4	5	8	5

attachment is by way of the dental cuticle, this calculus should strip easily from the tooth surface. Modes Two and Three would appear to fall in between these two extremes. Therefore it is easily understood why dentists have experienced such a great variance in the ease or difficulty of calculus removal. The fact that we deal most often with a combination of the described modes of attachment points to the importance of curettage rather than use of scalers.

Another observation made on the 50 specimens is pertinent to the periodontist. The cemento-enamel junction seems a favored place for a type of calculus formation which makes use of the anatomical

Fig. 4. Calculus attachment into cementum. (660×) A—Cementum B—Micro-organisms extending from calculus matrix into cementum. C—Calculus D—Dentin

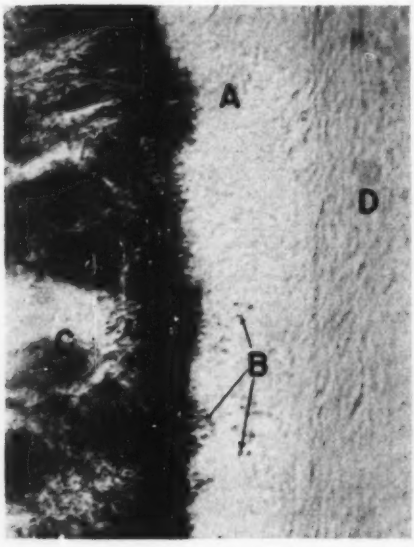
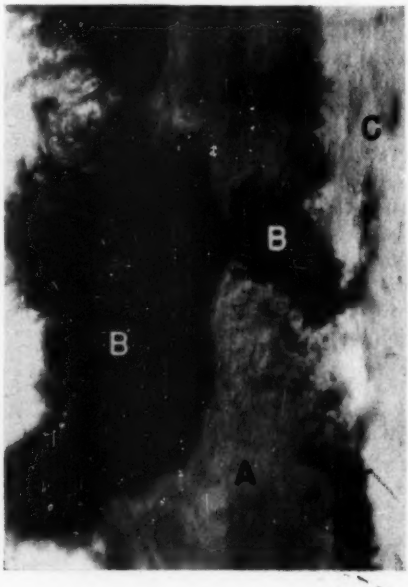


Fig. 5. Calculus attachment into area of cementum resorption (660×) A—Cementum B—Calculus C—Dentin. Micro-organisms of calculus matrix have penetrated through cementum and along dentino-cemental junction.



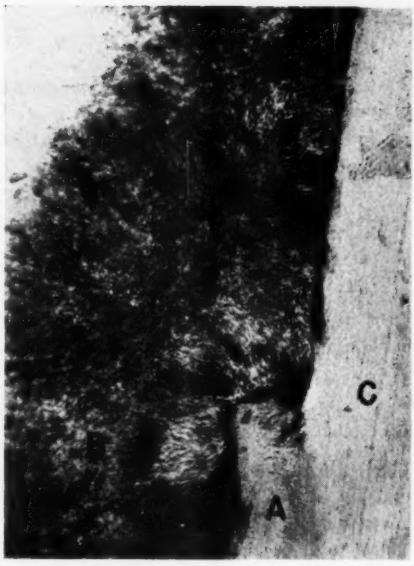


Fig. 6. Calculus at cemento-enamel junction. (280×)
A—Coronal portion of cementum B—Calculus
C—Dentin (no enamel cover at this point)
Calculus is attached to cementum and dentin. Micro-organisms have invaded both cementum and dentin and penetrated along cemento-dentinal junction.

irregularities found here. Often there exists a space between the cementum and the enamel so that dentin is exposed. This may have occurred during tooth formation and cementum or enamel may have been fractured and removed. In any event it makes an ideal spot for anchorage and attachment of the bacterial matrix of calculus. (Figure 6). A recent publication[6] calls attention to the fact that better clinical results are obtained when the cervical enamel, including the area of the cemento-enamel junction enamel and undoubtedly some of the cementum, is removed. The observation under the microscope supports this clinical impression.

SUMMARY AND CONCLUSIONS

Four modes for the attachment of the bacterial matrix of calculus to the root surface are recognized. This finding provides an explanation for the ease or difficulty of calculus removal and emphasizes the need for careful curettage.

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The Effect of Toothbrushing on the Keratinization of the Gingiva

by S. SIGMUND STAHL, D.D.S., M.S.; NATHAN WACHTEL, B.A., D.D.S.; CARLOS DECASTRO, D.D.S., AND GEORGE PELLETIER, D.D.S., *New York, N. Y.*

IT IS the common opinion of most dentists that gingival stimulation is helpful in maintaining optimum oral health.[1] It is thought that massage of the gingiva stimulates circulation in the connective tissue and aids in the keratinization of the epithelium.[2] Robinson and Kitchin[3] in their histologic investigation of the effect of toothbrushing on the gingiva, noted increasing keratinization of the surface epithelium following toothbrush massage over a period of two months.

Montgomery,[4] as well as Miller, Soberman and Stahl,[5] studied the keratinization of the gingiva by means of the Papanicolaou technique. They noted that the exfoliated cells of the gingiva showed a high degree of keratinization and that the proportion of keratinized to non-keratinized cells was fairly constant in the individuals examined. Due to the difficulty of obtaining biopsies for the study of massaging effects on the gingiva, an attempt was made to utilize exfoliative cytology in this problem. The effect of a soft nylon toothbrush as compared to an extra hard natural bristle brush on gingival keratinization was also studied.

MATERIAL AND METHOD:

A group of 10 males, students at New York University College of Dentistry, volunteered for this study. All of the students were in good health and their gingiva appeared clinically normal.

By means of a wooden tongue depressor gingival scrapings were taken from the labial surface of the lower anterior teeth. These scrapings were obtained twice weekly, with an interval of three days between the first and second scraping.

The material was immediately spread on a glass slide, immersed in equal parts of alcohol and ether and processed according to the technique outlined by Papanicolaou.[6]

A random count of 100 cells was made for each slide and the cells were classified into three groups:

1—blue staining cells with nuclei,
2—red staining cells with nuclei and
3—yellow staining cells with pyknotic nuclei or anuclear.

The percentage of yellow staining cells in the count was used as an index of keratinization.

The study was divided into three periods:

The first period lasted two weeks and included four scrapings. The students were not instructed in any special technique of brushing, but were merely told to continue in their normal manner.

The second period lasted four weeks and included eight scrapings. At the beginning of this period, the students were given a soft nylon brush and were instructed to continue in their normal brushing technique.

The third period lasted four weeks and included eight scrapings. During this period, the students were given an extra hard natural bristle brush and again continued brushing in their normal manner.*

FINDINGS:

The cell types observed in this study were similar to those observed by Montgomery[7] and Miller, Soberman and Stahl.[8]

The yellow cell count was taken as an indicator of the degree of keratinization of the gingiva and the following average yellow cell count was noted:

2 week preliminary period—60 yellow cell/average

4 week soft nylon brush—58 yellow cell/average

4 week hard natural brush—69 yellow cell/average.

The complete figures obtained from this study were then subjected to statistical analysis and revealed that:

1—No significant difference exists between the degree of keratinization noted during the preliminary period and the soft brush period.

2—Significant difference exists between the degree of keratinization noted during the soft brush period as compared with the hard brush period.

The technique employed was not accurate enough to detect daily changes in keratinization. The use of a greater number of individuals is necessary to obtain such detailed changes.

COMMENT:

The findings in this study confirm clinical as well as histologic evidence, namely that toothbrushing can increase keratinization of the gingiva. Since greater gingival keratinization may afford relatively greater protection against injury,[9] the use of massage should be of benefit in maintaining oral health.

An attempt was also made in this study to evaluate the effectiveness of the newer, multitufted soft nylon brushes against the hard natural bristle brush. It appeared, from this experience, that greater keratinization results from the use of a hard brush in clinically normal appearing mouths. However, this does not preclude the use of the softer brushes in certain select periodontal conditions.

SUMMARY AND CONCLUSIONS:

10 male dental students were utilized in a study of the effect of toothbrushing on the keratinization of the gingiva. The experimental period lasted 10 weeks. During this time, a soft multitufted nylon brush and an extra hard natural bristle brush were used. The degree of keratinization was estimated according to the average number of exfoliating keratinized cells.

The following results were noted:

1—Gingival keratinization increased with increased massage.

2—In normal mouths, a hard natural bristle brush caused greater keratinization than a soft multitufted nylon brush.

3—The use of oral scrapings seemed well suited to the study of changes in keratinization following changes of local environment.

Department of Periodontia, New York University College of Dentistry.

*The brushes were obtained through the courtesy of the Pycopé Co., Jersey City, New Jersey.

The authors wish to express their thanks to Mr. Jules Joskow, Department of Economics, City College of New York, for his statistical analysis of the data and to Dr. Samuel Charles Miller, chairman of the Department of Periodontia, New York University College of Dentistry, for his help in the preparation of this paper.

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Biopsy Studies of Necrotizing Ulcerative Gingivitis*

by ERWIN M. SCHAFFER, D.D.S., M.S.D., *Minneapolis, Minnesota*

ALTHOUGH a number of reports have been published on both the bacteriological and histo-pathological aspects of necrotizing ulcerative gingivitis, they have been contradictory. Based upon a study of a case of biopsied ulcerative membranous gingivitis, Tunnicliff[1] reported that both fusiform bacilli and spirilla were present in the necrotic tissue and were invading living tissue, with the spirilla being considerably in advance of bacilli. No cocci were observed in the living tissue. Cahn[2] also found a great many fusiform bacilli but scarcely any spirochetes penetrating the tissue for quite a depth adjacent to the tooth. The case biopsied had been treated for ten days and was asymptomatic. The gingiva appeared to be normal. However, Stammers[3] found that the organisms were only in the superficial layers of living tissue but not in the deeper tissue. Box did not find them in the deep vital tissues.

Apparently there is no unanimity of agreement on the presence of bacteria and the histo-pathologly in lesions of necrotizing ulcerative gingivitis. Therefore, it was felt that a study of biopsies from a large number of patients with a clinical diagnosis of necrotizing ulcerative gingivitis would be worthwhile.

EXPERIMENTAL PROCEDURE

Biopsies were taken in 36 patients who had typical lesions of necrotizing ulcerative gingivitis. They were also taken from 12 control patients who were in the same age group as the experimental. In these control patients, no gingivitis was visible and the gingivae appeared "normal." Under local anesthesia, the interdental papillae were excised with a scalpel making two vertical incisions from the area of the gingival margin downward to the bone on the labial and one horizontally from the labial to the lingual just superior to the crest of the alveolar bone connecting the two vertical incisions (Figures 1, 2 and 7). With a blunt curette the papillae were removed. The tissue was fixed with Zenker's solution, washed, embeded in paraffin, sectioned, and stained with Gram Weigert's bacterial stain. Some of the sections were also stained with Levaditi's stain.

RESULTS

In the 36 experimental biopsies, bacteria were found on the surface as follows: pleomorphic spirochetes on 10, pleomorphic fusiform bacilli on 8, cocci on 27, bacilli (other than fusiformis) on 5, and filamenting types on 6 (Figure 3). No vibrios were seen but fibrin and debris was a common finding. A part of every section was ulcerated (Figure 4). In other parts of the sections the epithelium was intact and all layers could be distinguished. As the intact epithelium approaches the denuded ulcerated area one can see that the stratum corneum and all or part of the stratum granulosum has disappeared (Figure 4). The keratinous layer was present on the epithelium in 21 cases; however, it decreases in thickness toward the ulcerous lesion. Acanthosis, a slight distance from the margins of the ulcers, was observed in 20 and edema in 30 of the biopsies. The epithelium near the ulcer was infiltrated with mononuclear leukocytes in 17 of the biopsies and with polymorphonuclear leukocytes in 11 (Figure 5). No bacteria were

*From Part II of a thesis submitted in partial fulfillment of the requirements for the degree Master of Science in Dentistry at the University of Minnesota. This project was directed by Dr. Raymond E. Johnson and Dr. W. J. Simon of the School of Dentistry and Dr. Raymond N. Bieter of the School of Medicine.

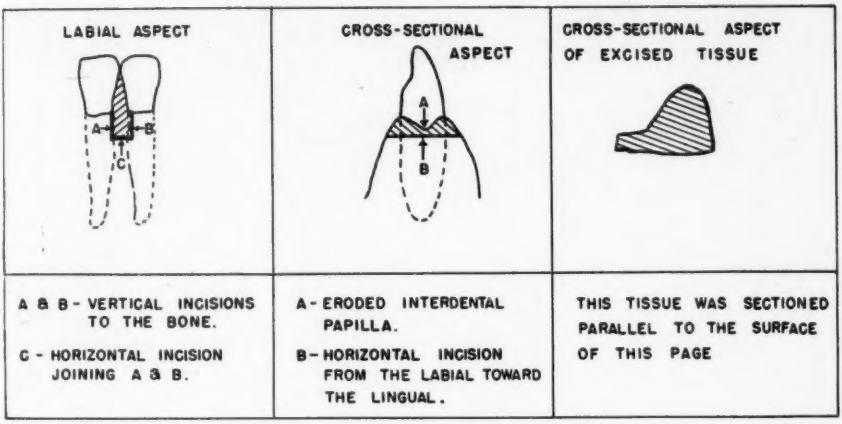
LABIAL ASPECT
A
B
C
CROSS-SECTIONAL ASPECT
A
B
CROSS-SECTIONAL ASPECT OF EXCISED TISSUE
A & B - VERTICAL INCISIONS TO THE BONE.
C - HORIZONTAL INCISION JOINING A & B.
A - ERODED INTERDENTAL PAPILLA.
B - HORIZONTAL INCISION FROM THE LABIAL TOWARD THE LINGUAL.
THIS TISSUE WAS SECTIONED PARALLEL TO THE SURFACE OF THIS PAGE

Fig. 1. Diagram illustrating Biopsy Excision.

found in the inter and intra cellular areas of the epithelium. The area devoid of epithelium consisted of necrotic tissue which was infiltrated with inflammatory cells, both plasma cells and lymphocytes (Figure 4). This occurred in all cases but polymorphonuclear leukocytes occurred in only 11 of the cases. Another common finding was the presence of erythrocytes outside the vessels in various degrees of edema. Bacteria were seen on and in the necrotic tissue but never in the deeper layers of the vital tissues.

In the 12 control biopsies the epithelium was continuous in all except one. No ulceration and necrosis of epithelium was present (Figure 6). Keratin was found covering the epithelium in all biopsies. It was partly absent in only two. Acanthosis was seen in two biopsies. In no case was the epithelium infiltrated by bacteria and leukocytes but lymphocytes and plasma cells were seen in the connective tissue in all of the biopsies. There were bacteria on the surface of the epithelium, but they were apparently fewer in number than in the sections of necrotizing ulcerative gingivitis. Cocci were found in 6 and bacilli (other than fusiformis) on 1. No spirochetes, vibrios or filaments were found.

Fig. 2. Biopsy excision of interdental papilla between the lower left second bicuspid and first molar.

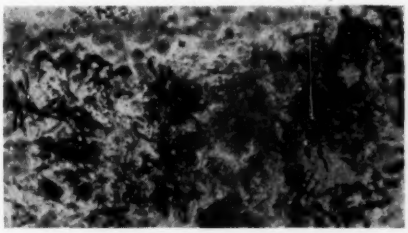

Fig. 3. Biopsy showing pleomorphic bacteria on the surface of a necrotizing ulcerating gingival lesion. ×680.

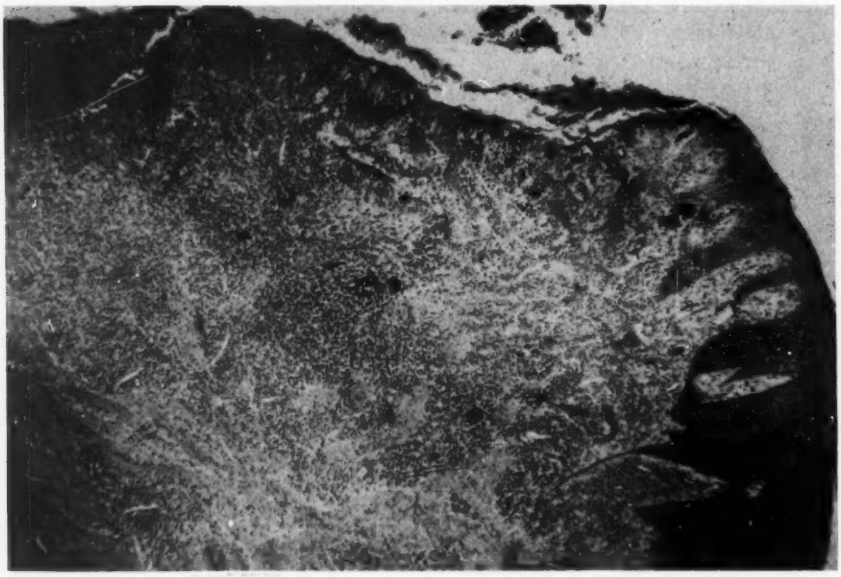

Fig. 4. Biopsy of necrotizing ulcerative gingivitis. Adjacent to the ulcer the epithelum is intact. The connective tissue is infiltrated with inflammatory cells. ×36.

DISCUSSION

The important difference between the biopsies from the "normal" and the necrotizing ulcerative gingivae was the presence of an area devoid of epithelium in the necrotic gingivitis cases. The clinical appearance of a punched-out (cupped, crater-like papillae) is really a gradual process of erosion of the keratin, stratum granulosum, and on down until all of the layers of the epithelium have disappeared. The presence of inflammatory cells in both the experimental and control tissue demonstrates again that this is not necessarily a differential characteristic. Micro-organisms apparently are found not only on the surface but also in the necrotic areas, while in the controls they were only on the surface.

The presence of leukocytes in the epithelium could only be demonstrated in the biopsies of necrotizing ulcerative gingivitis and not in the controls.

In all of the patients the excised gingivae regenerated in 14 to 21 days without any complications such as secondary infection, scarring, or recession. After three weeks it was impossible to see any difference between the biopsied area and the other affected interdental papillae. The healing after biopsy is illustrated in Figure 7. The healing of treated patients was the same in the experimental as in the controls.

SUMMARY AND CONCLUSIONS

In this study, 36 biopsies were taken from 36 patients with necrotizing ulcerative gingivitis. Control biopsies were taken from 12 patients.

In the sections from necrotizing ulcerative gingivitis, the pathological process appears to begin on the surface of the epithelium. First, the keratin is destroyed, then the degeneration progresses through the various layers of the epithelium including the basal layer and continues on into the immediate fibrous tissues. The adjacent tissues become edematous and infiltrated

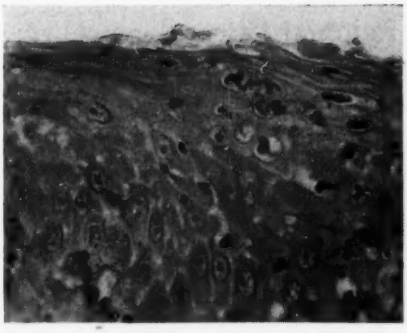

Fig. 5. Mononuclear and polymorphonuclear leukocytes infiltrating the epithelium adjacent to the ulcer. Higher magnification of Figure 4. ×680.

Fig. 7. Healing of the interdental papilla between the lower left central incisor and the right central incisor 12 days after excision. The healing 9 days after excision can be seen between the lower left central and lateral incisor. The papilla between the lower left lateral and cuspid has just been excised to study healing and the effects of treatment.

with inflammatory cells, so that necrosis continues laterally and more deeply, if unchecked. The bacteria apparently do not penetrate vital tissues but infiltrate the tissues as they become necrotic. Possibly the toxins destroy the tissues first.

Ulceration, necrosis, sloughing, and erosion was found in all of the lesions, but in the controls they were absent except for erosion in one section.

Fig. 6. Biopsy from a so-called "normal" gingiva. Keratin is present on the intact epithelium. There is no ulcer.

Because of these findings the biopsy can be used as an aid in the differential diagnosis of necrotizing ulcerative gingivitis.

In this study there were no complications from the biopsy excisions; because of this and the findings in this study, the biopsy can be used as an aid in the differential diagnosis of necrotizing ulcerative gingivitis.

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ACKNOWLEDGMENT

Illustrations by the photographic library of the School of Dentistry University of Minnesota.

Reattachment in Periodontal Disease

by MAX SHAPIRO, D.D.S., *Beverly Hills, California*

REATTACHMENT of the soft tissues to the tooth, with or without bone regeneration, has been observed clinically as far back as 1893 by Allport[1] and 1894 by Younger.[2] Leonard[3] and others[4-10, 17] have reported clinical evidence of reattachment. Box[11] and Beube[4] have shown histologic confirmation of reattachment in human material.

Glickman and Lazansky[5] have demonstrated when doing gingivectomy on dogs, that it is possible for epithelium to attach itself to a higher crownward level than the point of incision. Linghorne and O'Connel[7, 8] observed reattachment of connective tissue and the formation of new bone and cementum in surgically created defects in dogs. Ramfjord[6] has demonstrated reattachment histologically in pathologic pockets experimentally produced in monkeys.

Moreover, Beube[4] and Ramfjord[6] have shown that where pocket measurements indicated clinical reattachment, histologic examination of this same material showed the formation of new bone, periodontal membrane, cementum, and epithelial reattachment at a more crownward level.

Nevertheless, there is a general feeling that reattachment can seldom if ever be achieved under routine periodontal therapy.[12, 13, 14, 15, 16]

The following clinical investigation was designed to determine the extent and frequency of clinical reattachment under carefully controlled conditions on randomly selected patients having periodontal disease with pocket formation.

As part of the diagnosis the following was recorded: Full-mouth roentgenograms, study models, pocket contours around the teeth, gingival condition, occlusal trauma, tooth mobility, food impaction, dietary intake.

METHODS OF MEASUREMENT

In order to be able to ultimately differentiate between reattachment and gingival recession following treatment, the depth of the pockets were determined from a fixed point (incisal edge, defect, or other markings) on the tooth.

A steel probe (Merritt) calibrated in millimeters was inserted to the full depth of the pocket and the distance to the gingival margin noted and recorded (distance B in Figure 1). Hydrocolloid impressions were taken of the mouth at the same time in order to have a permanent record of the teeth and gingival contours, before and after treatment.

EXPERIMENTAL

As a routine procedure, prior to treatment, all patients were referred to a physician and/or laboratory for physical examination including serum calcium, phosphorus, cholesterol, blood sugar, basal metabolic rate and/or blood iodine, complete blood count and urinalysis. Where defects were found treatment was instituted.

In addition, defects in occlusion were corrected.

All pockets were treated essentially in the following manner:

1. Under local anesthesia (block or infiltration into the muco-buccal fold), one quadrant of the mouth was treated to remove all calculus, using hoes, files and/or curettes.

2. To increase the time of epithelialization from the remaining gingival tissues and allow for organization of the clot within the pocket before epithelium enters pocket area, the gingival tissue was removed for a distance of ¼ to ⅓ the depth of the pocket at an angle. (Figure 2)

3. The soft tissue lining the remaining

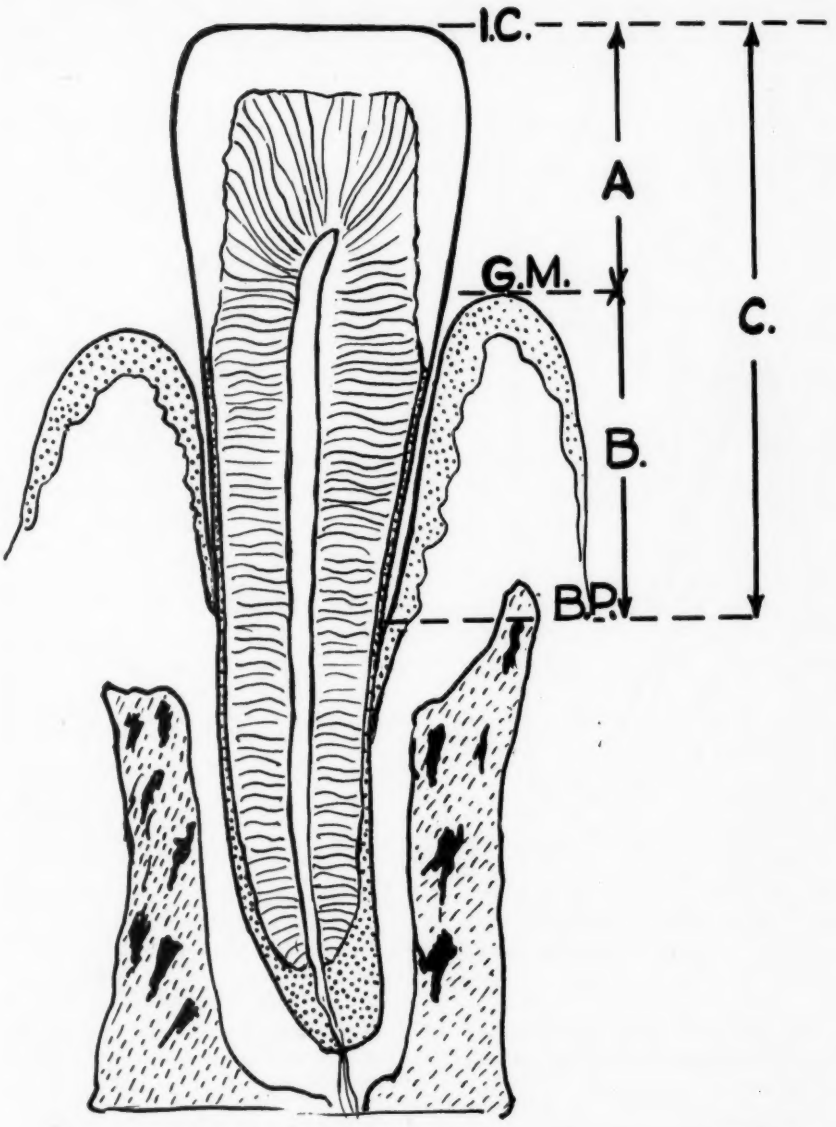

Fig. 1. I.C. Incisal edge (reference point). G.M. Gingival margin. B.P. Bottom of pocket.

depth of the pocket was curetted, and the base of the pocket penetrated to an arbitrary distance until considerable resistance was felt. This was done using curettes of the Younger-Good or Gracey type.

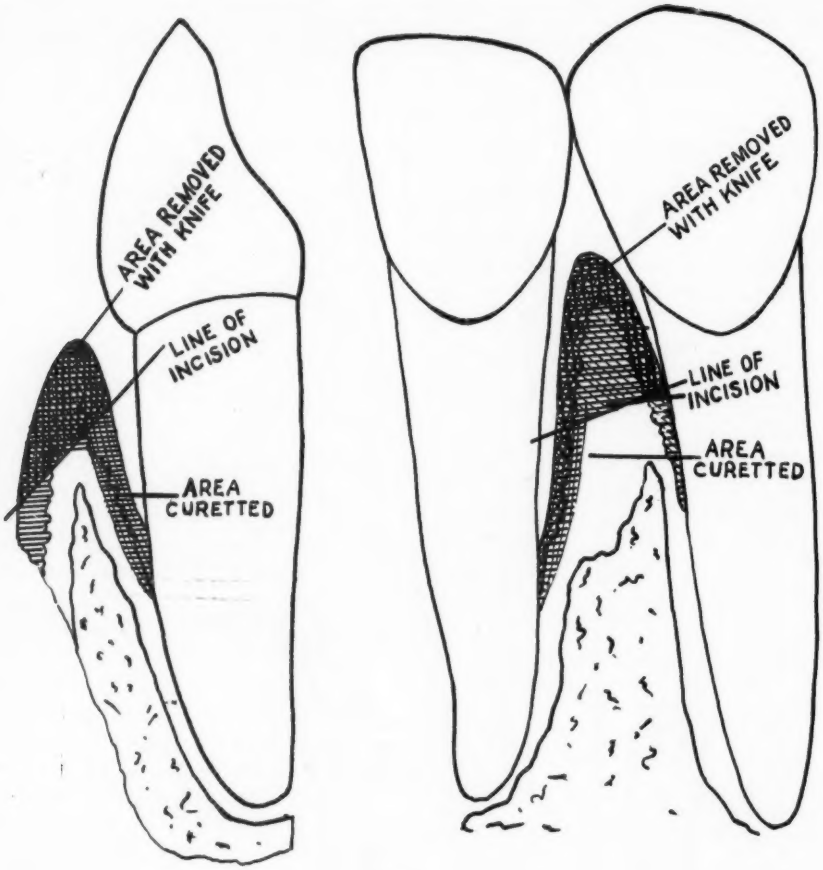

Fig. 2

From Proximal From Labial

Schematic drawing showing angle and position of initial incision.

4. No medicaments were used and the clot was permitted to form without further interference.

5. Whenever necessary, to reduce mobility, temporary splinting of wire or cast loops was applied.

6. A soft periodontal pack (modified Box's) was carefully applied. This pack was changed at the end of one week and removed at the end of the second week.

The remainder of the mouth was similarly treated, in quadrants, at one or two week intervals.

The teeth were polished and the patients taught proper home care using a modified Charter's technique.

No further therapy was done.

After an interval varying from three months to two years, the deepest pocket areas in twenty randomly selected patients

Patient # and sex	Age	Tooth & surface	Reattachment Pocket depth (millimeters)		Pocket base to reference point (A & B in Fig. 1)		Reattachment (millimeters)
A	B	C	D		E	F	G
			Before	*After*	*Before*	*After*	
1. Mr. A. B. #134 M.	43	⌈2 Dist.	4	2	12	10	2
2. Mrs. H. W. #163 F.	39	3 Mes.	7	4	14	12	2
		7 Mes.	6	3	12	10	2
		3 Dist.	5	1	10	7	3
3. Mrs. F. A. #94 F.	56	4 Buc.	9	6	12	9	3
4. Mrs. J. C. #145 F.	44	3 Dist.	5	3	9	8	1
5. Mrs. E. S. #157 F.	36	⌊4 Ling.	10	5	17	11	6
		4 Mes.	10	5	15	11	4
6. Mrs. R. L. #103 F.	38	3 Mes.	8	4	14	8	6
		2 Mes.	5	2	13	8	5
7. Mrs. J. S. #116 F.	46	7 Dist.	7	4	11	11	0
8. Mrs. C. G. #126 F.	48	7 Mes.	5	2	9	8	1
		6 Mes.	5	2	10	8	2
		5 Dist.	6	4	11	10	1
9. Dr. H. C. #107 M.	41	⌊2 Mes.	9	2	16	10	6
		⌊3 Dist.	10	4	19	12	7
		5 Dist.	7	3	10	8	2
10. Mr. A. S. #93 M	49	5 Dist.	7	3	13	10	3
		4 Ling.	8	3	14	10	4
11. Mrs. J. E. #98 F.	39	⌊6 Mes.	9	3	12	7	5
12. Mrs. B. F. #152 F.	47	1 Mes.	7	3	14	11	3
		2 Dist.	6	2	13	9	4
		6 Mes.	9	3	13	8	5
13. Mrs. D. S. #158 F.	35	⌊2 Mes.	6	3	12	10	2
		⌊1 Dist.	5	3	11	10	1
14. Mr. D. M. #47 M.	47	2 Mes.	6	2	13	10	3
15. Mr. J. S. #167 M	48	6 Dist.	6	4	11	10	1
16. Miss J. G. #74 F.	37	⌊4 Mes.	9	4	13	11	2
17. Mrs. F. I. #124 F.	24	⌈2 Dist.	5	3	10	8	2
		⌈3 Mes.	5	3	10	8	2
18. Miss R. S. #169 F.	48	⌊1 Ling.	10	3	18	11	7
		⌊1 Dist.	11	5	18	13	5
		⌊2 Mes.	10	2	16	10	6
19. Mrs. W. P. #148 F.	39	⌊2 Dist.	7	3	14	12	2
20. Mr. A. B. #179 M.	42	⌈2 Dist.	7	3	15	12	3
		⌈2 Mes.	9	5	17	14	3

(out of a group of two hundred) were remeasured.

At this time, the pocket depth was measured from the base to the gingival margin (Distance B, Figure 1). To this was added the distance from the gingival margin (at exit of probe) to reference point on tooth which was visible on patient and original study model (Distance A, Figure 1). Distance A on original model at this time was added to recorded original pocket depth.

RESULTS

Table 1 shows a summary of the collected pertinent data. Column E and F show the changes in position of the base of the pocket in relationship to the reference point which were obtained by measuring the base of the pocket to the gingival margin (Distance B, Figure 1) and adding to this the distance from point of exit of probe to a fixed reference point on tooth (Distance A, Figure 1). Column D shows the actual pocket depth before and after treatment. This is included only as a minor point of interest to indicate severity of original condition, and the status at the time of final measurement. It must be emphasized that the reattachment is only indicated by changes in the distance from base of pocket to reference point (Column E and F).

Column G tabulates the reattachment in millimeters within the individual pockets before and after treatment. The values are obtained by subtracting the distances in Column F from distances in Column E.

It can be seen that the extent of reattachment was variable, but in almost all cases there was some degree of reattachment. Figure 3 shows a bar graph illustrating the distribution of the extent of reattachment.

DISCUSSION

The nature of the reattachment in the above experiment is not known; however the work of Ramfjord[6] and Beube,[4] etc.,

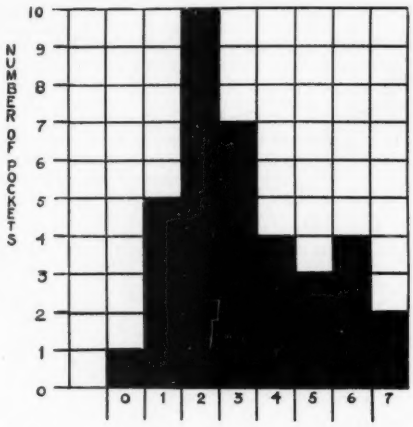

Fig. 3. Extent of reattachment (millimeters).

indicates that these changes which occurred may be a result of the formation of new connective tissue, bone, and cementum, as well as epithelium which reattaches at a higher level.

This will have to be demonstrated by further investigations; however roentgenographic evidence in this same series of cases indicates a considerable degree of new bone formation, the report of which is now in the process of preparation.

Ramfjord has indicated a possible error of ± .5 mm. in his study involving similar measurements. For this study, if the error is considered to be ± .5 mm., then the possible number of pockets showing reattachment would be in the group of 2 mm. or over (Figure 3), which compose 30 out of 36 or 83%. If the experimental error under these more difficult conditions of measurement is considered to be ± 1. mm. then the possible number of pockets showing reattachment would be in the group of 3 mm. or over, which comprise 20 out of 36 or 65%.

These findings indicate that the problem of reattachment should be carefully reconsidered.

SUMMARY

Preliminary evidence indicates that with the operative procedures described above, reattachment occurred to a variable degree in at least 65%, and possibly 83% out of 36 pockets measured, in 20 randomly sampled patients.

The author is indebted to Dr. Norman S. Simmons of U.C.L.A. in the preparation of the manuscript.

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THE AMERICAN INSTITUTE OF DENTAL MEDICINE ORGANIZED

At the Ninth Annual Seminar for the Study and Practice of Dental Medicine held in Palm Springs, California, October 26, 1952, the Board of Directors announced the founding of "THE AMERICAN INSTITUTE OF DENTAL MEDICINE." This Institute is non-profit in character and is so incorporated under the laws of the State of California. Its prime purpose is to further postgraduate education in the field of Dental Medicine. The Annual Seminar will continue as in the past 9 years as one of the essential functions of this Institute. In addition its members will be offered a case history each month from the broad field of Dental Medicine. These histories will be accompanied by original 35 mm. Kodachromes of the oral condition, pertinent laboratory findings, medical background, roentgenograms, histologic slides, photomicrographs, or whatever may be indicated in each individual case. This service will begin January 1953. Any member of the ADA or AMA and their foreign equivalents are eligible for membership. Further information may be obtained from the Executive Secretary of the Institute, Miss M. Lewis, 2240 Channing Way, Berkeley 4, California.

White Folded Gingivo-Stomatosis*

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Portland, Oregon

CASE NO. 1 (R.M.)

In August 1949 there presented at the Dental School of the University of Oregon a 23 years old white male patient, (R.M.), a mail carrier, for diagnosis of an unusual oral condition. The mucosa of the cheeks offered a whitish, soft, thickened and folded appearance (Figs. 1 and 2). The lesions looked as if the tissue had been parboiled. The vestibular surface of the lips appeared somewhat similar but less severely affected (Figs. 3 and 4). Closer observation revealed some vesicles within the limits of the affected area, and desquamation of the surface epithelium (Fig. 2). The lesion terminated at the muco-gingival junction approximately, leaving the gingivae practically unaffected (Fig. 5). The patient presented a folded tongue (Fig. 6); the underside of the tongue showed unusual horizontal striations (Fig. 7). The palate was free from grossly observable pathology. The condition was painless except for a burning sensation whenever the patient consumed acid and highly seasoned foods.

During 1949 and 1950 this patient received a thorough physical check-up, and routine dental operative and periodontal care. Within this period the lesions varied in intensity from time to time as to both appearance and clinical symptoms. In order to avoid irritation, the patient was advised to eliminate smoking and the consumption of alcohol and spices; he was also told to avoid food and drink of very high temperature. In addition he was instructed in proper oral hygiene and was prescibed a mild alkaline dentifrice.

The patient was born in Charles City, Iowa, in 1926. Neither parent is said to have been afflicted with a similar oral anomaly, nor is it known to have occurred among uncles, aunts, or grandparents. The patient's mother is living and well. His father died at the age of 49 of carcinoma of the lungs. Several more cases of cancer have occurred on either parental side of the family. The patient is married and has two children, a boy (4 years) and a girl (2 years), both living and well. His wife and the two children are reported free of any lesions of the oral mucosa. Nor did one brother, born 1924, who was killed in action in 1944, have comparable oral lesions, as far as known. The patient's only living sibling, a brother (D.C.M.), suffers from an oral condition of a character similar to that observed in the present case. The brother's oral picture will be described later in this paper.

Our first patient, R.M., had the common childhood diseases. The anomaly of his oral mucosa is said to have been first observed about the eleventh year of his life during an illness accompanied by high temperature. In 1939 he was a patient at the University of Iowa Hospital in Iowa City for treatment of nervous and physical exhaustion. The diagnosis in the Hospital Records at that time was that of "post-influenzal asthenia" and "congenital anomaly of the oral mucous membrane." A biopsy of the oral mucosa was taken in Iowa at that time and described as follows: "Marked increase in the thickness of the epidermis with scattered round cell infiltration just beneath the basal layer. The section is normal in all other respects." Diagnosis: "Hyperkeratosis of the buccal mucosa, probably congenital." According to the Iowa Hospital Records, the oral condition

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*This study received financial support from the Oregon State Board of Dental Examiners.

Fig. 1. R. M. (Case No. 1). Left buccal mucosa showing folded, whitish and thickened appearance.

had been increasing in severity for the two preceding years. The buccal mucosa was described as thickened, fissured and white.

The patient did not remember any appreciable change in the condition of his oral mucosa in the ten intervening years after his release from the Iowa Hospital. At the time of our first examination of the case (August 1949), the following additional data were obtained. No major diseases in adulthood. No history of tuberculosis or lues; serology (Kahn), repeated three times, was always negative. The eyes were normal. Physical examination of nose and throat was negative while a thickening was found in the tympanic membranes. On anteroposterior roentgenograms of the head the frontal sinuses appeared congenitally undersized. The patient also complained of a persistent sense of fullness in the ears; he felt "as if the contents were forced into the ears, when he blew his nose." There was a two-year-old lesion of dermatophytosis on the palm of the right hand and some desquamation between the toes; no other abnormalities existed on the skin, particularly on nails and palms of hand and feet, elbows and knees. The texture of the skin appeared normally soft. Chest and abdomen showed no pathological change. Proctoscopic examination was negative. The patient was shown to have a transitional first sacral vertebra, a congenital anomaly consisting of a supernumerary underdeveloped vertebra located between lumbar and sacral segments of the vertebral column. Upper and lower ex-

Fig. 2. R. M. (Case No. 1). Right buccal mucosa; appearance same as in Fig. 1. Note also vesiculation (a) and desquamation (b).

tremities were normal. Red, white and differential blood counts, sedimentation rate and glucose tolerance were found within normal limits. No pathology was discernible in a routine urine analysis. The patient's intelligence appeared normal. Saliva was sparse and ropey, oral hygiene only fair. The patient complained of a scum depositing on the teeth, particularly noticeable on arising in the morning. Intraoral full mouth roentgenograms failed to reveal any periapical, or sizable periodontal pathology. Crops of lesions of herpes labialis were reported by the patient to occur rather frequently.

During the years of 1947 to 1950 this patient was intermittently under the care of the Veteran's Hospital in Portland, Oregon. There he was treated for his oral lesions and aural complaints in turn with auto-vaccine prepared from bacteria of his nasal mucosa, with poly-vitamin preparations, benadryl, neosynephrine, and with repeated penicillin injections, receiving 14 administrations of 300,000 I.U. of this drug I.M. withhin two weeks. None of these treatments brought any improvement in the appearance of the oral lesions or the aural symptoms.

Fig. 3. R. M. (Case No. 1). Vestibulum of lower lip. Note striations and fields.

Fig. 4. R. M. (Case No. 1). Note the fields in the vestibular mucosa of the upper lip.

After receiving attention at the Dental School during the school term of 1949-1950, the patient left town and was not seen by us again until May, 1952, almost three years after he had been observed by us for the first time. No appreciable changes in the appearance of his oral lesions or his symptoms related to the ear could be noticed.

A small piece of the afflicted mucosa of the cheek was removed for histologic examination in April, 1950, and again in May, 1952. The histologic picture in the later

specimen was the same as in the one taken two years earlier. The sections were stained with H & E and with Wolbach's[1] modification of the Giemsa stain. A few preparations were also made with Wilder's[2] silver reticulum stain, and some were stained for keratohyalin, following the method described in Carleton.[3]

Findings in the specimen taken in 1950 follow: The sections stained with H and E show parakeratosis in some fields (Fig. 8). Hornification is absent in the remainder of the specimen (Fig. 9), as is usual in the mucosa of the cheek. From the most superficial layers to the germinative layer the epithelium appears considerably thickened

Fig. 6. R. M. (Case No. 1). The patient has a folded tongue with crenated border.

Fig. 5. R. M. (Case No. 1). The lesion can be seen to stop at the muco-gingival junction in the upper jaw.

in total diameter, and acanthotic in places (Fig. 9). No clear differentiation into a stratum spinosum and granulosum can be made. Many of the cells of these two strata are hydropically degenerated; many of the nuclei appear pyknotic, and quite a few cells lack any nucleus even in the deeper layers (Fig. 8, 10). The cells throughout this large area are bizarre in size and shape.

Cytoplasmic, extranuclear, basophilic granules can be seen in the more superficial, but also in the deeper layers of the stratum granulosum and spinosum; they are easily visible in the H & E stained preparations and in the Giemsa stained sections (Fig. 11). When stained for keratohyalin these granules appear dark, and must therefore be considered to be keratohyalin. In the deep layers of the stratum spinosum, adjoining the stratum germinativum, spaces can be seen either within the prickle cell layer, or separating the prickle cell layer from the germinative layer. These spaces are filled with a serous exudate and are somewhat reminiscent of the intra-epithelial vesicles in herpes simplex[4] and of the lacunae in Darier's disease.[5] In the area of the stratum

Fig. 7. R. M. (Case No. 1) Horizontal striations can be seen at the underside of the tongue.

Fig. 8. R. M. (Case No. 1). Parakeratosis (A) is seen in some fields. H & E Magnification 180.

spinosum adjoining the stratum germinativum can be noted an increased incidence of pyknosis and vacuolization (Fig. 10). The stratum germinativum appears to consist of two to three cell layers and is everywhere sharply demarcated against the more superficial epithelial layer. It shows an increased number of mitoses; these are, however, of a normal type. In several areas there appears a separation of cells of the stratum germinativum from the overlying epithelium for some distance. In some spots it seems that almost the entire epithelium has separated from the tunica propria; however, even in these areas a few cells of the germinative layer have remained adhering to the underlying layer. The observed vesiculation can therefore be considered strictly intra-epithelial. The basement membrane seems well defined and intact in the specimen stained with Wilder's silver stain (Fig. 12). None of the changes in the epithelium described is of malignant nature.

In the tunica propria the papillary layer shows inflammatory cellular infiltration following the contour of the germinative layer and also arranged in perivascular fashion (Figs. 9, 10). The cytologic nature of this exudate is mixed polymorphonuclear neutrophilic and round cell in character, the proportions varying from field to field, with the former outnumbering the latter over all. There is also a considerable number of fibroblasts in the papillary layer. In the deeper parts of the papillary layer and in the reticular layer fiber bundles predominate and only few connective tissue cells are apparent. These fiber bundles seem homogenized and broken up in places, so as to give the impression of scarring or perhaps of hyalin degeneration.

In the reticular layer are also seen vascular spaces, some of which contain polymorphonuclear neutrophiles in numbers larger than usual. Some few round cells can be seen here also in the perivascular fields. The predominance of the polymorphonuclear neutrophiles in view of the chronicity of the condition points to acute exacerbation due to repeated irritations from the defects in the epithelium.

A remarkable large number of mast cells can be seen in the tunica propria of sections stained with Wolbach's modification of the Giemsa stain. Some of their granules appear scattered within the connective tissue, a phenomenon observed occasionally where mast cells occur.[6] There are also present in the tunica propria a number of basophilic polymorphonuclear leucocytes (Fig. 13). The function of these two last named cell types is not well understood.

CASE NO. 2 (D.C.M.)

The second case is that of a brother (D.C.M.) of the patient described above. He was born in Marble Rock, Iowa, in 1923 and was first seen by us in March, 1952. This patient is also married and has three children, a girl (5 years), and two boys (2 years and 1 year). Wife and children are reported to be free of any comparable oral lesions. Just like his brother, this patient is a mail carrier by occupation. He has had the common childhood diseases and no major disease in adulthood. There is no history of tuberculosis or syphilis.

The appearance of his oral mucosa resembles closely that of his brother. The patient first noticed the oral changes in 1936, when 13 years of age, after a long illness during which his temperature rose to 102 degrees F., and during which he was confined to bed for six weeks. Ever since, the mouth lesion has persisted; localization and clinical appearance (Fig. 14), while slightly less pronounced, are similar to those shown by his brother. However, in the second patient (D.C.M.) the lesion exceeds the muco-gingival junction in the lower jaw; there is a papillary gingival hyperplasia with the mucosa showing heavy stipling (Fig. 15). On this picture can also be seen a vesicle and an area of desquamation in the region of the attached and free gingiva about the lower right cuspid. Here, just like in his brother, intra-oral full mouth roentgenograms fail to reveal any periapical, or sizable periodontal pathology. The patient appears to be in good health. Off and on he has been in treatment for a persistent irritation of the nasal sinuses. The patient feels that the oral lesion has remained more or less unchanged in the last 16 years. He reports that occasionally a fine kernel is dislodged from the mucous membrane of the mouth and states that he is not greatly bothered by his oral condition. The patient's pulse rate is slow (58); his BMR is plus 5 and was zero when taken in 1937. Red, white and differential blood count, serology (Kahn) and urine analysis fail to reveal any pathology. No eosinophiles are found in the differential blood count. A nasal smear shows only 4 eosinophiles to 1000 cells. A proctoscopic examination gives negative findings. The complete physical check-up fails to reveal any further pathology, especially any anomaly of skin or skin appendages. In this patient as well as in his brother (R.M.), number, size, shape and clinical appearance of the teeth are normal.

A biopsy specimen was obtained from the mucosa of the left cheek. The histologic picture resembles closely the one described in the preceding case of his brother.

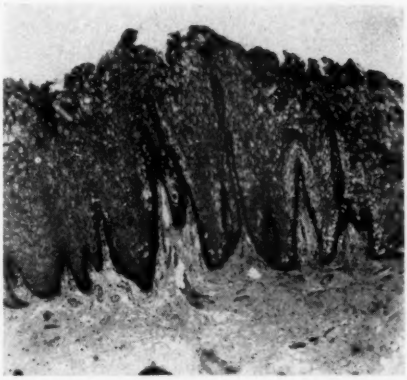


Fig. 9. R. M. (Case No. 1). In other areas keratinization is absent; there is acanthosis and thickening in the epithelial diameter. H & E Magnification 100.

DISCUSSION

Several possibilities were considered in the interpretation of these two cases. One might feel on the basis of the epithelial changes (intra-epithelial vesiculation, hydropic changes, and presence of cytoplasmic inclusions) and of the exudative infiltration in the propria that one were dealing with an infectious inflammatory condition, possibly of virus origin.

An allergic etiology has been considered by one of our consultants and by the physician at the Veterans Hospital in Portland, Oregon, to exist in case No. 1 (R.M.); this view was later abandoned as unlikely because of the normal number of eosinophiles in the blood (4% of the white count in R.M., 0% in D.C.M.), because of the insignificance in numbers of eosinophiles counted in the nasal smears (zero per 1000 cells in R.M. and 4 per 1000 in D.C.M.), and lastly in view of the fruitlessness of anti-histaminic therapy.

There are a very few clinical and histologic features in the condition that are slightly reminiscent of desquamative gingivo-stomatitis (gingivosis).[7-9] The desquamation of the epithelium, when observed under the microscope in our cases, differs from that seen in desquamative stomatitis

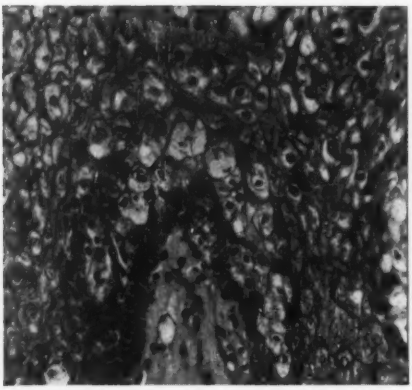


Fig. 10. R. M. (Case No. 1). Note the hydropic degeneration of the cells of the stratum granulosum and spinosum. Many nuclei are pyknotic and many cells lack any nucleus. The reticular layer shows round cell infiltration. H & E Magnification 400.

in that in our cases the separation occurs within the epithelium and not between epithelium and propria as in desquamative stomatitis.[8] Our sections also show a well developed basement membrane, a structure generally absent in desquamative stomatitis.

Vitamin A deficiency can probably be ruled out as etiologic factors on the basis of the fact that the two brothers live in

Fig. 11. R. M. (Case No. 1). In all layers of the stratum granulosum and spinosum are seen basophilic, cytoplasmic, extra-nuclear granules; they take the keratohyalin stain. H & E Magnification 1000.

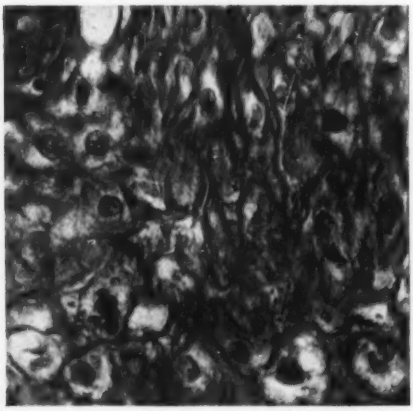
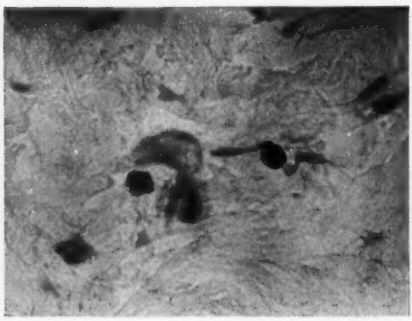


Fig. 12. R. M. (Case No. 1). Basement membrane (a) intact. Tunica propria (b). Intraepithelial vesicle at (c). Wilder's stain. Magnification 186.

different households and are the only afflicted members of their families, and again from the lack of success of Vitamin therapy.

We explain the apparent vesiculation in the absence of both typical balloon cells and intranuclear acidophilic inclusion bodies as indicative of dyskeratosis, not of a virus infection. Hydropic degeneration is entirely compatible with dyskeratosis.[10] The premature pyknosis and loss of nuclei observed even in the deeper layers of the stratum spinosum are apparently caused by a disturbance in the maturation of the epithelium. We believe that in the absence of uniformity the extranuclear cytoplasmic

Fig. 13. R. M. (Case No. 1). Basophilic granulocytes in the propria. Giemsa stain. Magnification 1650.



basophilic granules can not be considered of viral etiology, but that they are keratohyalin granules, a view supported by their appearance in keratohyalin-stained sections. The irregular distribution of these keratohyalin granules in the stratum granulosum and spinosum give further strength to the diagnosis of dyskeratosis postulated above. We feel that the changes in the membrana propria, consisting of hyalinization of the fiberbundles in the reticular layer and of leucocytic infiltration are secondary and caused by long-continued chronic irritation.

There is a certain similarity to Darier's disease in the histologic picture. One might here consider D.M.'s statement that he feels sometimes as if some kernel were coming loose in his mouth. However, we do not consider Darier's disease the exact diagnosis because of the absence of the typical skin lesions clinically, and because of the absence of corps ronds in any of the biopsy specimens studied histologically.

The variety of lesions of skin and mucous membranes occurring in ectodermal dysplasias is very great.[11] Certain features observed in our two patients have also been described by other writers describing their cases of ectodermal malformations. Anomalies of the respiratory system for instance have often been reported in dysplasias of the outer germ layer,[12,13] and changes in the tympanic membrane are mentioned in a case of generalized congenital keratoderma.[14] White lines at the underside of the tongue have been reported in a case of pachyonychia congenita.[15] Brünauer in reporting on generalized congenital dyskeratoses[16] describes cases presenting deep folds in the mucosa of the cheeks; he also mentions the gradual appearance and development of the lesions, an interesting statement in view of the history of our two patients who first observed their lesions in early adolescence. Again, the frequent occurrence of herpes labialis and of hyperhidrosis, observed in our patients, has been stressed in comparable lesions.[17] All the cases mentioned that showed similar pathology of the mucous membrane, presented also extensive abnormalities of the skin. Cannon's "White Sponge Nevus of the Mucosa"[18] differs from our cases in that this author describes the lesion to have existed since infancy, and as covering not

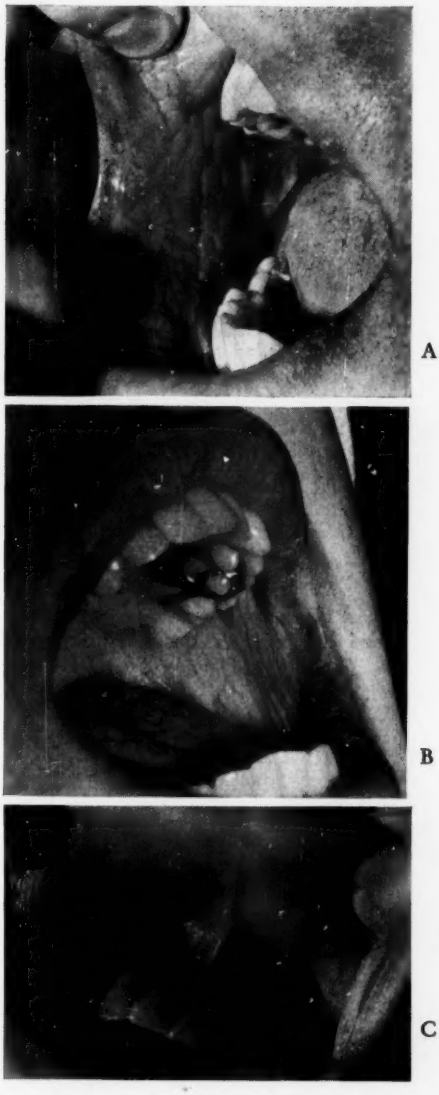


Fig. 14. D. C. M. (Case No. 2). (A) Right cheek. (B) Left cheek. (C) Vestibulum of upper lip.

only cheeks and vestibulum, but also floor of the mouth and both hard and soft palate. The gingiva was entirely free in Cannon's cases, while it was involved in one of ours. No vesiculation is described as present in Cannon's patients, while it occurred in our cases. In his paper are also mentioned "numerous small, clear, follicular openings, ranging in size from a pinpoint to that of a pinhead, scattered over the surface," a feature absent in our cases. The patients described by Cannon are a grandmother and her four adult children, one of whom has an adolescent son; all these members of the family are shown to be afflicted with the anomaly. In two of the cases, where that author had occasion to examine rectum and vulva, he found their condition practically identical with that of the mucosa of the mouth, while in our cases the rectum was found free from any lesions.

There is one report in the literature presenting a similar malformation of the oral mucosa as the sole manifestation of ectodermal dysplasia. In Ludy and Shirazy's case, described as "Congenital Leukokeratosis Mucosae Oris,"[19] the family history revealed the patient's mother, her maternal aunt, and her two sisters to have been afflicted similarly. This report differs in the following points from ours. These authors describe a "villous" appearance of the affected mucosa which we were unable to notice in our cases. Their lesion involved also the palate and the anterior pillars of the fauces, regions appearing normal in our patients, who in turn showed white striations on the underside of their tongue. In one of our patients (D.C.M.) the lesion involved also the gingiva, a region reported free in Ludy and Shirazy's case, and in both brothers vesiculation could be demonstrated in some parts of the affected oral area, a phenomenon not observed by Ludy and Shirazy. In the single case described by those authors the anomaly had been present since birth, while our two cases were first noticed in early adolescence, supposedly following an attack of a prolonged febrile condition. Furthermore, both our patients showed some pathology of the

Fig. 15. D. C. M. (Case No. 2). Papillary hyperplasia with heavy stipling (A). Vesiculation and desquamation in the area of attached and free gingiva (B) about lower right cuspid.

respiratory system and their father died of carcinoma of the lungs. One of the brothers, D.C.M., also complained of repeated attacks of herpes labialis. The other one, R.M., had an additional anomaly of the mesoderm, a supernumerary vertebra in the lumbosacral segment. This is not an uncommon malformation,[20] but of interest here because of the occasional co-existence of anomalies in the number of vertebrae and visceral abnormalities.[21] The histologic changes observed in our sections seem much more severe than either in Cannon's,[18] or in Ludy and Shirazy's[19] cases, but, like theirs, are definitely not malignant. The mode of inheritance is dominant in both Cannon's, and in Ludy and Shirazy's case reports; the same might be assumed in our two patients.

SUMMARY AND CONCLUSIONS

Cases of two brothers are described showing a folded, whitish and soft oral mucosa of cheeks and vestibular surfaces of the lips, involving also the underside of the tongue and, at least in one patient, affecting also the gingiva. There are also vesiculation and desquamation in isolated parts of this area. The condition has existed since early adolescence; no anomalies of the skin are demonstrable. In view of the clinical appearance, history, and histologic examination, the lesions are classed as belonging in the group of congenital ectodermal dysplasias. On the basis of the folded, whitish appearance of the lesions and their chronic, partly degenerative character we

suggest the designation "White Folded Gingivo-Stomatosis."

ACKNOWLEDGMENTS

The authors wish to express their appreciation for the assistance they have received from Dr. Ellis B. Jump, Professor of Anatomy, University of Oregon Dental School, in the preparation of the photomicrograms, and from Dr. Jeff Minckler, Associate Professor of Pathology, University of Oregon Dental School, for consultation. We also wish to acknowledge with thanks the help we have received from the Regional Office of the Veteran's Administration and the Hospital of the Veteran's Administration, both of Portland, Oregon.

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DR. ORBAN RECEIVES HONORS

Dr. Balint Orban of Colorado Springs, Colorado, was given the Miller prize at the XIth International Dental Congress in London. The prize is given by the Federation Dentaire Internationale every five years "for outstanding achievemets in the field of international dentistry." Dr. Orban is Director of the Colorado Dental Foundation in Colorado Springs.

Readers will also be interested to know that Dr. Orban has accepted the position of Professor of Periodontics of the Periodontia Department of Loyola University School of Dentistry in Chicago. He will spend two weeks out of every two months in Chicago. He will be assisted by Dr. Frank M. Wentz, D.D.S., M.S., Ph.D., as Associate Professor.

The Apparent Antagonism Between Caries and Periodontal Disease*

by Basil G. Bibby, *Rochester, N. Y.*

THE inclusion of the word "apparent" in the title of this paper is significant. This is so because when one attempts to establish the realities of the relationship between periodontal disease and dental caries, the question as to whether there is a genuine antagonism between the two conditions remains unsettled. The fact that someone such as I, whose investigations have centered in the problem of dental caries, should be called upon to discuss this subject indicates that there is a dearth of authorities in this field and that there must be very few clinicians or research workers who have been able to make serious studies of both dental caries and periodontal disease. It is understandable that those dental practitioners who are primarily concerned with restorative dentistry should be inclined to overlook mild periodontal involvements but have vivid recollections of the striking cases in which advanced periodontal pathology has demanded the sacrifice of teeth with sound or satisfactorily restored crowns. Conversely, periodontologists generally see patients whose teeth have survived the ravages of dental decay and whose teeth are sufficiently sound to justify periodontal treatments, and thus, their experiences also tend to strengthen the idea that periodontal disease and caries do not occur in the same mouths. The perspective of the research worker is no better. In these days of specialized research, in which the investigator is largely the slave of his specialized techniques, his experiences can be more narrowing than the clinician's, and as a result his conclusions are liable to be biased by his original impressions or ideas. In any case, it should be noted that in periodontal studies the laboratory worker has to depend upon others, in this case clinicians, to provide him with starting material for his investigations. Laboratory studies can never be better than the clinical studies on which they are based. This point is so important that it will be reiterated at the end of this paper.

With this preliminary warning that opinions on the antagonism between periodontal disease and dental caries may have resulted from limited observations, we can proceed to an examination of some of the evidence which is available on the subject. First, however, it should be noted that until recently any attempt to correlate periodontal disease with other conditions has been greatly handicapped by the absence of any accepted basis of nomenclature or classification of various types of periodontal involvements. While this situation has improved, it is still hazardous to draw definite conclusions from published work unless satisfactory descriptions of the clinical conditions under study have been provided. Fortunately, in spite of differences in the recording methods used, the assessment of caries activity presents less of a problem. Hence, our principal difficulty is to avoid unnecessary confusion between various periodontal states. Obviously, the simplest division of periodontal states will be safest for the purpose of this review. Therefore, as far as possible, conditions recognizable as gingivitis, periodontitis and periodontosis, as the terms are used by the Academy of Periodontology, will be discussed separately in this report.

Of the few papers which have attempted to bring together the evidence relating to the possible antagonism between periodontal disease and dental caries, the most recent are those of Kesel[1] and Maldonado.[2] The former review refers to some clinical reports but emphasizes the laboratory studies and concludes that the weight of

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TABLE I

GINGIVITIS AND CARIES IN SCHOOL CHILDREN

(Adapted from Brucker)

	Number Examined		*Average No. Cavities Per Child*	
	Boys	*Girls*	*Boys*	*Girls*
Healthy Gingivae	735	757	2.5	3.3
Slight Gingivitis	72	47	3.3	3.7
Severe Gingivitis	12	11	4.0	5.0

evidence points to an antagonism between periodontal disease and dental decay. Maldonado who supports a review of biochemical findings with his own clinical surveys decides that there is a high activity of caries with gingivitis and periodontitis complex but a relative caries immunity in association with periodontosis.

In attempting a further analysis on this subject, we propose to examine first of all the *epidemiological literature* to determine whether published work indicates any type of relationship between the incidence of caries and gingivitis, periodontitis or periodontosis. Next, we will attempt to evaluate the existing knowledge of the *systemic or physiological factors* associated with these types of periodontal disease to see if there is reason to believe such factors would modify resistance to dental caries. Thereafter, we will consider the effects of *local factors in the mouth* such as calculus or saliva to see whether the available evidence indicates that they might promote antagonistic tendencies between caries, on the one hand, and gingivitis, periodontitis or periodontosis, on the other.

EPIDEMIOLOGICAL STUDIES

It has already been pointed out that the idea that periodontal disease and caries are antagonistic, springs from clinical observations, and it has also been suggested that these clinical impressions may be deceptive. Because it is all too easy for the human mind to be influenced by preconceived ideas, it is necessary to compile figures on the occurrence of disease or the results of therapy if reliable conclusions are to be drawn as to the effects of associated conditions. Such epidemiological data is not plentiful in regard to the relationships between periodontal disease and dental caries but such of it as seems reliable will be reviewed here.

GINGIVITIS

There seems to be more direct or indirect evidence relating gingivitis to caries activity than is the case with other types of periodontal pathology. In one study, Brucker[3] found that 735 boys and 757 girls with healthy gingiva had an average of 2.5 and 3.3 cavities per child; that 72 boys and 47 girls with slight gingivitis had 3.3 and 3.7 cavities and that 12 boys and 11 girls with severe gingivitis averaged 4.0 and 5.0 cavities (Table I). He also noted that there seemed to be less gingivitis in colored children than in white children. Since the former are less prone to dental caries, he thereby added some support to his general conclusion. Another study which compared the activity of dental decay with the occurrence of gingivitis in military personnel was made by Shay and Smart.[4] They found gingivitis most frequently in patients with no fillings and decreasing occurrence as the number of fillings increased. There was more gingivitis in patients with untreated caries than in a corresponding group of

TABLE II

GINGIVITIS AND FILLINGS IN MILITARY PERSONNEL

(Adapted from Shay and Smart)

Gums	*Fillings*				*Untreated Caries*	
	0	1-2	3-5	6	*Absent*	*Present*
Normal	35	35	43	40	40	11
Gingivitis	56	49	35	31	25	22
% Gingivitis	60	58	45	44	48	67
		P = .04			P = .02	

"normal" patients. This was taken to indicate a lower than average caries susceptibility in association with gingivitis. Their conclusions were sanctified by statistical analysis and are summarized in Table II. Maldonado[2] found more than half the teeth carious in 11 gingivitis patients which was a much higher incidence than found in periodontosis or "periodontitis with atrophy." He felt that caries was very active in gingivitis patients (Table III). Rosenthal[5] found lower lactobacillus counts in 50 gingivitis patients. The University of Rochester Research Team carried out studies on the incidence of caries and gingivitis in Puerto Rico and the Virgin Islands. The relationship between these conditions was not distinct. In Puerto Rico there was less caries and more gingivitis than in the Virgin Islands. It was suggested[6] that the lower incidence of gingivitis in the Virgin Islands group might be due to the negroid character of the children examined.

Indirect evidence regarding the relationship between the occurrence of gingivitis and caries can be deduced from other publications such as the excellent review of Marshall-Day[6] on the epidemiology of periodontal disease. He presents data on the occurrence of gingivitis in the United States, Great Britain, the Isle of Lewis, India, New Zealand, Puerto Rico and the Virgin Islands. Because the criteria used by the 10 different investigators cited varied considerably, comparisons of their findings are hazardous. However, the breadth of the variations found in the incidence of gingivitis (2.2 to 99.4%) and the fact that a number of the studies were made by the same groups of examiners leaves no doubt that wide variations in the incidence of gingivitis occur in different parts of the world. These variations do not parallel differences in the incidence of dental caries. For instance, while dental caries in Rochester, New York is considerably more active than in New Jersey, the figures quoted for gingivitis incidence are respectively 73% and 8.8%. Also, in Marshall-Day's[7] own studies, the poorly nourished children in the Kangra district of India had a very low activity of dental decay and an 81% incidence of gingivitis, whereas in the higher socio-economic group in Lahore with a relatively high caries activity, gingivitis was found in only 73% of the children. Similarly, the relatively caries free Maori children in New Zealand showed 94% gingivitis whereas an active caries group in Rochester had 73%, and a group with intermediate caries activity in Great Britain presented the lowest figure with a 60% incidence of gingivitis. Brucker[3] has also noted divergence between caries and gingivitis figures in different localities pointing out that studies in Atlanta, Georgia and New South Wales reveal a predominance of caries over gingivitis while studies in Tristan da Cunha and Manus Island, South Africa and Australia

TABLE III

INCIDENCE OF CARIES IN DIFFERENT PERIODONTAL INVOLVEMENTS
(Adapted from Maldonado)

	No. of Patients	*No. of Teeth*	*% of Teeth with Caries*
Periodontosis	12	353	8%
Periodontitis with Atrophy	27	611	14%
Periodontitis with Horizontal Atrophy	37	974	50%
Gingivitis	11	274	55%

show a preponderance of gingivitis over caries. The most convincing evidence in this area is in the four studies in which Marshall-Day participated for which the percentage of children with gingivitis is shown in Table IV. The lowest incidence of caries (India) is associated with highest gingivitis and the highest incidence of caries (Virgin Islands) with the least gingivitis. From these and other studies, Marshall-Day[7] concluded that gingivitis and caries vary independently of each other.

Because compilations of the number of cavities in the teeth are a record of past caries activity rather than existing activity whereas observations on gingivitis record only the active state of the disease, it is important to keep in mind that observations on gingivitis made at one time may be different from those made shortly thereafter. Indeed some of the studies at the University of Rochester[8,9] have indicated considerable variations in the gingival picture over short periods. Thus, none of the data so far presented really casts any light on the question of whether the causal agencies of caries and gingivitis operate in opposition to each other. To determine the extent to which the two diseases progress simultaneously or have dissimilar causation, repeated long range studies are needed. Reports of such studies cannot be found in the literature. However, some information bearing on this question can be obtained by comparing the amount of new caries and gingivitis occurring at different ages. Using Marshall-Day's[6] age curve for gingivitis and Moore's[10] data for caries, such a comparison has been made (Chart I). From the data presented, it seems obvious that the factors initiating dental caries and gingivitis are active at different ages and may, therefore, be assumed to be distinct.

Other indirect evidence of different causation of gingivitis and dental caries can be found in the war-time experience in concentration camps and displaced persons. The findings of King et al.[11] in Gibraltar and Schultz-Haudt[12] in Norway demonstrate that conditions which give rise to a dramatically increased occurrence of gingivitis caused an almost complete inhibition of caries activity.

The only conclusion that seems possible from the divergent data which has been offered is that while there is some evidence in groups of the same composition to indicate an increase of dental caries with gingivitis this evidence, which is based on small numbers of gingivitis patients, is not convincing. On the other hand, there are a number of studies which indicate that factors such as race, geography, dietary habits, hygienic practices or inadequacy of diet influence gingivitis independently of caries. Although some of these factors seem to work in opposite directions, there is not adequate proof that there is direct antagonism between this type of periodontal disease and dental caries.

TABLE IV

PERCENTAGE OF CHILDREN WITH GINGIVAL DISEASE IN RELATION TO CARIES
(Adapted from Marshall-Day)

	India	*Virgin Islands*	*Rochester, N. Y.*	*Puerto Rico*
Age Group	*249 Subjects Low Caries*	*823 Subjects High Caries*	*234 Subjects Intermediate Caries*	*1488 Subjects Intermediate Caries*
12	73	64	81	79
14	82	66	75	71
16	74	55	73	65

PERIODONTITIS

The epidemiological information dealing with the association between the more advanced forms of periodontal involvement, the so-called periodontitis, and dental caries is of much the same order as that dealing with gingivitis. It leaves similar doubt as to the nature of the relationship between the two conditions. Several reports by Black,[13] Marshall-Day,[6] and Chilton[14] present evidence on the occurrence and distribution of periodontal involvements involving bone destruction. The first suggestion of an antagonistic relationship with dental caries seems to have been made by Pickerill[15] who stated in 1914 that pyorrhea can be considered as the pathological antithesis of dental caries. Cahn[16] believed that pyorrhetic teeth were resistant to dental caries. These ideas do not seem to have been supported by statistical information. The first report in which we have been able to find statistical data is that of Tishler[17] who examined 5,000 teeth in 200 patients in which he found 2,675 cavities. He concluded that there was no antagonism between caries and periodontal conditions with "pocket formation and suppuration." Similarly, Maldonado[2] found dental caries in 50% of the teeth he examined in 37 patients with "paradentitis." This he considered to be evidence of high caries activity. Miller and Seidler[18] examined 1,003 patients, found about the same incidence of caries and noted that 62% of the males and 56% of the females required periodontal therapy. Indices of periodontal disease were worked out by determining the amount of bone destruction in X-rays. These were compared with caries scores calculated according to the Bodecker Index. Males were found to be more susceptible to periodontal disease and females more susceptible to dental caries but no clear relationship could be demonstrated between periodontal disease and caries in individual patients. Lactobacillus counts were used by Rosenthal[5] as a means of indicating the nature of the relationship between caries and "pyorrhea." The finding of lower counts in a group of 50 "periodontal" patients was taken as an indication of an opposing relationship. In general, the evidence cited above does not indicate that dental caries and periodontitis are antagonistic. However, it should be realized that the conditions examined represent the cumulative effects of the diseases and do not necessarily represent the forces which were working during their progressive stages. In other words, the possibility remains that in their active phases periodontitis and caries may inhibit each other, but either may at one time or another progress actively and leave a permanent record of that activity in the patient. Support for this possibility is found in Miller and Seidler's[19] study of the relative susceptibility of different teeth to "alveoloclasia" according to age. They found that

maxillary and mandibular first and second premolars and the mandibular third molars were least susceptible; that first and second molars were next in order and that maxillary and mandibular first and second incisors and first, second and third molars had the highest susceptibility to periodontal disease. Marshall-Day[6] has recorded similar findings. Although the authors drew no comparisons with caries activity in their studies, it is interesting to note that there is great dissimilarity when their findings are compared with Klein's[20] graph for caries susceptibility of the various teeth (Chart II).

The age occurrence of periodontitis may also have some bearing on its relationship to dental caries. Miller and Seidler[18] showed bone destruction beginning in 9% of the 11 to 15 year old hospitalized patients which increased steadily until 97% of the patients were affected in the 41 to 45 age group. Marshall-Day[21] has reported comparable observations. Since dental caries affects 90% or more of the children in the 11 to 15 year age group in the United States and slows down before or during the third decade, it is fairly obvious that there is a difference in the age activity of dental caries and periodontitis.

On the basis of the evidence offered in this section, it seems apparent that, in so far as the overall statistical studies are concerned, there is no distinct antagonism between periodontitis and dental caries. However, when the incidence of the disease in different tooth types is considered, there is distinct indication that the conditions which produce caries do not produce periodontal disease. Were this the case, we would expect caries and periodontal destruction to occur in the same teeth and not in contrasting tooth groups as has been observed. Similarly, if these conditions were caused by the same systemic or local factors, we should expect them to occur at the same time in life because the evidence cited shows that this is not the case. In short, although there is no convincing evidence that the conditions are antagonistic, it is fairly apparent that they depend on the different causative agencies.

PERIODONTOSIS

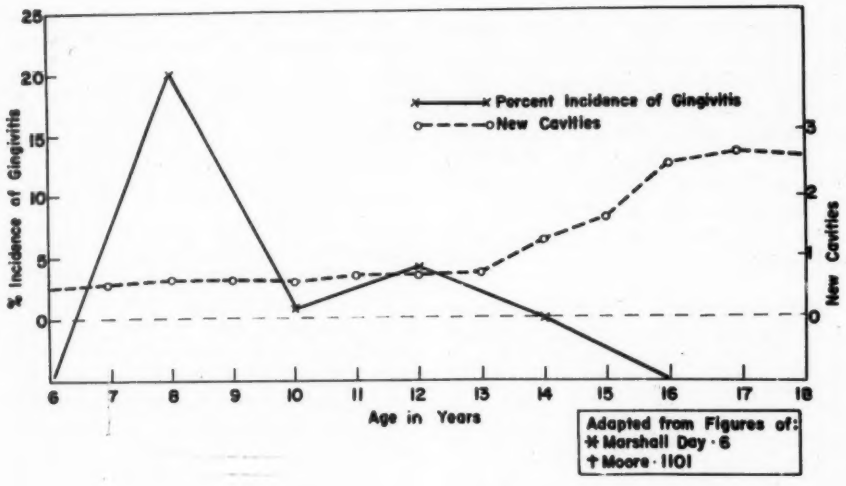
The likelihood of an antagonistic relationship between periodontosis and dental caries has been subject to less question than is the case with other types of periodontal involvements. This, however, is partly the result of the small amount of critical study which has been focused on this possible relationship. Most of the ideas dealing with it rest on opinion rather than fact. Maldonado[2] quotes the work of Lang and a number of other investigators in the support of this idea, but cites no actual epidemiological data other than his own. Actually, Maldonado's study seems to contain the only quotable epidemiological evidence in this area (Table III). In 12 periodontosis patients he found only 8% of the teeth with dental decay. This is less than 1/6 of the percentage showing dental decay in mouths of his gingivitis or periodontitis groups. Rosenthal[5] reported lower lactobacillus counts in a group of 50 periodontosis patients than in a similar control group. Since Maldonado studied only a small group of patients and Rosenthal depended upon lactobacillus counts to estimate caries, neither of the studies justify drawing a definite conclusion. The most that can be claimed regarding a possible antagonism between caries and periodontosis is that limited clinical evidence indicates the existence of such a relationship.

PHYSIOLOGIC AND SYSTEMIC RELATIONSHIPS BETWEEN DENTAL CARIES AND PERIODONTAL DISEASE

A great deal of theorizing has been indulged in concerning the systemic causation of periodontal disease. As with ideas on the systemic background of dental caries, theory has gone ahead more rapidly than the acquisition of facts. Although a large number of laboratory studies have been made on blood and other body fluids of periodontal patients, the uncertainty as to the actual type of periodontal involve-

CHART I

Percentage of New Cases of Gingivitis* and New Cavities† from 6-18 Years



ment studied reduces the interpretative value of some of these, and none have been correlated directly with caries activity.

GINGIVITIS

Indirect evidence that systemic factors which predispose to gingivitis do not cause dental caries can be found in the previously mentioned clinical studies of King[11] and Schultz-Haudt[12] where gingivitis occurred on inadequate diets in association with a reduced activity of dental caries. Certain animal experimentation supports these findings. For instance, on the one hand, it has been possible to induce "extensive lesions of the gingiva and periodontal tissues" in vitamin C deficient monkeys[22] while on the other, there is no comparable evidence that vitamin C or other deficiencies predispose towards dental caries in animals or human subjects. Children on near starvation diets have been shown[7] to have a low activity of dental decay and high gingivitis.

PERIODONTITIS AND PERIODONTOSIS

Reviews of opinions on the subject of the relationship between caries and periodontal disease (involving bone destruction) have been offered by Miller and Seidler[18] and Maldonado.[2] Note must also be taken of Broderick's[23] claim that in caries there was a tendency toward sympathicotonia producing acidosis and lymphocytosis whereas in periodontal disease there vagotonia associated with an increased blood alkalinity and eosinophila. Broderick did not support his claims with significant analytical data. Citron is quoted[1] in contradiction and is reported to have found diminished blood alkalinity in periodontal disease. Maldonado's review[2] deals with Broderick's claims in some detail and quotes the studies of Cabello[24] as supporting Broderick's hypothesis in so far as periodontosis is concerned but not for periodontitis.

Analytical studies on blood by 17 different groups of investigators have been

listed by Tenenbaum[25] and include determinations of cholesterol, uric acid, phosphatase, vitamin C, carbon dioxide combining power, sugar in the blood. Tenenbaum's own study of 70 "periodontoclasia" patients led him to conclude that the deviations from normal blood were slight and not consistent. Similar lack of consistency has been noted in comparisons of blood chemistry of patients with high and low caries activity.[26] Neither was Karshan[27] able to establish differences in blood chemistry associated with dental caries.

The most comprehensive study dealing with physiological and systemic influences on periodontal disease is that of Karshan.[28] This investigation on 30 patients with advanced marginal periodontitis, 59 with periodontitis complex, 77 with periodontosis and 51 control patients, embraced more than thirty biochemical tests on blood urine and saliva and included dietary analyses and physical examinations. Small deviations were found between the control and each of the periodontal groups in the blood level of vitamin C, glucose, globulin, albumin, calcium ion, alkaline phosphatase, red blood cell sedimentation, red blood count, white blood count and neutrophiles. No appreciable differences were found between the control and experimental groups for total calcium, inorganic phosphate, sodium, chloride, total protein, total cholesterol, free cholesterol, non-protein nitrogen, urea, uric acid, carbon dioxide capacity, hemoglobin, vitamin A and carotene, although deviations from the normal ranges were obtained for both groups. The Kline test was negative in all cases, and no differences were found in the basal metabolic rate. No subclinical deficiencies of thiamin, riboflavin or nicotinic acid were found in any of the subjects, but there was a low level of vitamin C in the plasma of many of the periodontosis patients. There were also inconsistent signs of a low urinary excretion of estrogen and of adrenocortical disfunction in some of the peridontosis patients. The physical examinations revealed no common systemic conditions. In short, no definite deviations from normal blood be established for the patients with various types of periodontal disease. In view of this, failure to produce evidence of physiological or systemic variations from normal in periodontal patients and the absence of known physiologic variations in dental caries, we must conclude as did Becks[29] that "the constitutional and internal factors which determine—the development of paradentosis—are as yet not wholly substantiated."

LOCAL FACTORS IN PERIODONTAL DISEASE AND DENTAL CARIES

It is now generally accepted that as in dental caries local factors are of primary importance in the initiation of certain types of periodontal disturbances. However, because of the vitality and responsiveness of the gingival tissues to systemic changes such as nutritional deficiencies or chemical poisonings, it seems inescapable that, unlike caries, the reaction of the gingiva to local effects is likely to be modified to a considerable extent by the systemic background. This would be true probably to a lesser extent of the other periodontal structures.

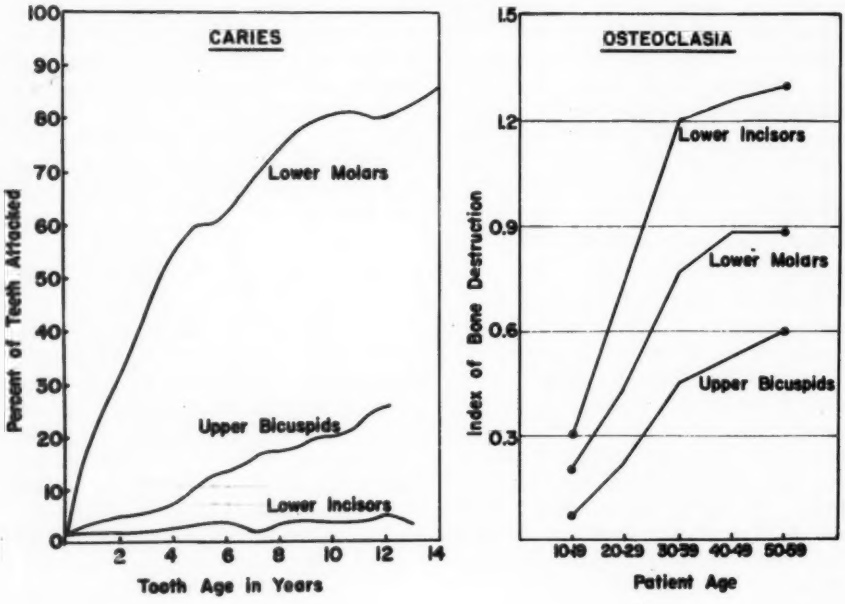
GINGIVITIS

The local factors predisposing to gingivitis are not well understood. The abundant personal experience of dental practitioners that marginal gingivitis occurs in dirty and unkept mouths with accumulations of debris around the gingival margins has received support in the epidemological survey of Brucker.[30] In a parallel study he offered evidence that there was more dental decay in children who never brush their teeth or do so seldom than in those who brush them daily. However, since the state of both the gingiva and teeth undoubtedly paralleled the level of the children's home care, one is not justified in assuming that the two diseases exerted an effect on one another.

Since gingival accumulations of bacterial debris are most generally associated

CHART II

Susceptibility to Caries and Periodontal Disease of Certain Teeth



with gingivitis, it is worth considering bacteriological associations with gingivitis on one hand and dental caries on the other. As far as the bacteriology of gingivitis is concerned, it has been suggested[31] that gingivitis is associated with superficial accumulations of mixed organisms which elaborate tissue irritating products causing inflammation with little bacterial penetration of the tissues. The bacteriological agents in dental caries are known to produce tooth decalcification by acid formed by fermentation of carbohydrates. Bacterial activity in accumulations of calculus may also contribute to gingivitis, but the other effects of calculus can be more appropriately considered in relation to periodontitis.

PERIODONTITIS

There does not seem to be any direct evidence as to whether the local factors causing or contributing to periodontitis oppose those conducive to dental caries. The complete absence of typical dental caries in periodontal pockets seems to indicate that the conditions provided by the pockets, if not those causing them, are not the same as those giving rise to dental decay. This would also seem to be true in so far as the formation of the subgingival or supragingival calculus are concerned for it is very rarely that calculus deposits and dental caries are found in the same locations on the same teeth. The only occasions which I have observed calculus depositions in caries lesions followed a radical change in diet and a complete inhibition of the caries activity.

There are theoretical reasons why the process of calculus formation and tooth decalcification are unlikely to occur on the same teeth or even the same mouth. Stated in the simplest way, it is not conceivable that the processes representing loss of cal-

cium from teeth and precipitation of calcium on the teeth from the saliva could occur simultaneously. Stated in a more impressive way, the solubility of hydroxyl apatite, which is the most important inorganic component of calculus, tooth enamel and dentine, is related to the pH of the saliva (Ericsson[32]) and the phosphate concentration of the medium in which it exists (Hardwick[33]). Since it has been shown[34] that the phosphate saturation of saliva from calculus forming mouths is higher than that of patients who do not form calculus, it can be concluded that saliva which would be likely to give rise to calculus (and periodontal disease) would not be prone to cause tooth decalcification (and dental caries). As far as subgingival calculus is concerned, the same basic physio-chemical laws undoubtedly apply, (Bibby[35]) but in this instance the source of the calcium phosphate for the calculus probably comes from tissue fluids as well as from the saliva. In general then it may be concluded that to the extent to which periodontal disease is dependent upon calculus formation it represents a process antagonistic to dental caries.

Related evidence as to the influence of saliva on the development of periodontal disease and caries has been offered by Karshan[26,27] who has made chemical studies of the saliva in both diseases. The stimulated and unstimulated saliva from caries-free patients had a higher calcium and inorganic phosphate content than that of caries-susceptible patients. The reverse was true of the periodontal patients in whose saliva both calcium and inorganic phosphate were lower in those with active periodontal disease. In this latter group, the pH of the stimulated saliva was also statistically higher. In addition Karshan[27] found that the carbon dioxide and calcium combining power was greater in caries free saliva, which property Leung[36] associates with calculus formation. Variations of a statistical sort have not been found in the many analyses for other salivary constituents which have been made on patients with or without periodontal disease or caries.

Observations relating to the proteolytic activity of saliva have indicated that antagonistic forces may be operating in caries and periodontal disease. Berg et al.[37] found that the saliva from patients with periodontal disease putrefies more rapidly than saliva from normal mouths, and it was suggested that the relationship between proteolytic capacity of saliva and periodontal disease is sufficiently reliable to have diagnostic value. That this might predicate reduced caries activity is suggested by Dreizen and Spies[38] demonstration that the end products of protein breakdown, such as indole, skatol, phenol and cresol, inhibit carbohydrate fermentation in saliva. Further evidence along these lines is the finding that ammonia forms more rapidly in individuals with periodontal disease (Kesel et al[39]). Berg et al[40] have suggested that because of a lowered glycogen content in inflamed or diseased periodontal tissues less acid formation is associated with it and the predisposition to caries is reduced.

It has also been shown[41] that glycolysis interferes with the de-amination of certain amino acids which suggests that conditions where there is much sugar fermentation, and associated dental caries, would not be favorable to the proteolysis and the related periodontal disease.

PERIODONTOSIS

A review of the writings on periodontosis does not reveal definite evidence of any variations in conditions of the oral environment which may be associated with it.

CONCLUDING COMMENT

In the nature of a general conclusion, it can be stated that the evidence indicating the existence of an antagonism between periodonta' disease and dental caries is not clear cut.

As far as gingivitis is concerned the clinical evidence fails to support this possibility and certain theoretical considerations, such as the higher incidence of gingivitis with dirty teeth, make it seem

possible that these conditions may sometimes result from a similar background. On the other hand, the increased occurrence of gingivitis in nutritionally deficient groups in association with a lowered activity of dental decay suggest that no such relationship exists. This apparently opposing evidence can be reconciled by assuming the existence of different types of gingivitis some of which are more dependent upon systemic than local factors. Those dependent upon systemic factors would seem to have a somewhat antagonistic relationship to caries.

As far as periodontitis is concerned the evidence of an antagonism to dental decay is partly supported by clinical evidence, and the difference between the time occurrence of this disease and its tooth to tooth distribution seem to suggest that it may be caused by opposing factors. Further evidence of a possible antagonism between the two diseases is found in an examination of local environmental factors. Calculus deposition and increased putrefaction associated with periodontal pathology oppose caries, and variations in salivary chemistry found in periodontitis occur with low caries activity.

In respect to periodontosis, clinical evidence of its relationship to caries is inadequate, but the available information indicates an antagonistic relationship. Although this condition is probably the result of physiological or systemic changes, thorough analyses of blood and saliva have failed to establish consistent deviation from normal. In this it resembles the situation with caries. Because there is next to no information concerning local factors in periodontosis, changes associated with the disease cannot be invoked to supply theoretical explanations as to how the possible antagonism to caries is brought about.

Finally, it should be noted that studies of periodontal disease of the sort we have reviewed indicate once again the need of thorough clinical study. It can't be emphasized too strongly that laboratory investigations cannot hope to solve clinical problems unless the refined laboratory methods which are available can be applied to groups of patients which are adequately differentiated on a clinical basis. In short, it is my belief that significant progress will not be made in solving periodontal problems until a more exact differentiation between types and reactions of various periodontal involvement have been established by painstaking clinical research.

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Book Review

THE GROWTH AND DEVELOPMENT OF THE NEGRO IN DENTISTRY IN THE UNITED STATES

Edited by CLIFTON ORRIN DUMMETT, D.D.S., *Chief, Dental Service, Veterans' Administration Hospital, Tuskegee, Alabama*

This interesting little volume of 124 pages, which has been published by the National Dental Association (printed by the Stanek Press, Chicago), represents the editing of the work of nine contributors on a subject about which little had been collected in the past. This report of the progress achieved by the Negro in dental medicine was begun in 1947 when the Editor was serving as the Dean of the School of Dentistry of Meharry Medical College.

The preface, prepared by Dr. Joseph F. Volker, Dean, School of Dentistry, University of Alabama, points out that there is an acute need for increased facilities for training Negro dentists and for the provision of funds for scholarships. In addition, opportunities for postgraduate and graduate study by Negro dentists and facilities for the education of Negro people to the need for dental services are other urgent demands. Dr. Dummett's book presents the background for these current needs by tracing Negro dentistry and dental hygiene from the records of the earliest American civilization through the post-emancipation period, the development of the National Dental Association and the present status of dental educational opportunity for the Negro.

An excellent chapter, entitled "The Negro in Dental Education and Specialization," provides objective data on (1) his undergraduate dental education, (2) his graduate education, teaching and research, (3) his specialization in dental practice, (4) his activity in public health dentistry and (5) his development of auxiliary dental personnel.

The public health worker, interested in the total problem of dental care, will find further data in this book on the supply and distribution of Negro dental personnel. He also will get a quick, illuminating view of interracial professional practices and relationships.

Kenneth A. Easlick, D.D.S., Professor of Dentistry,
University of Michigan, Ann Arbor, Michigan

Editorials

Dental Education and the Child

In the International Dental Journal[1] will be found a constructive and thought-provoking article bearing the above title. It is a plea for greater consideration to be given to prevention in dental education, not only as it affects the child but the adult as well. After reviewing at some length the history of dental education, attention is called to the report of the Curriculum Survey Committee in which it recommends that approximately one quarter of all undergraduate instruction be devoted to prosthetic dentistry, whereas the time allotted to the teaching of pedodontia, periodontia, and orthodontia is "quite insignificant." With a view to remedying this imbalance in dental education, it is recommended that "prosthetic dentistry already out of proportion with respect to time allotment . . . give way to preventive subjects."

Agreeing that whereas the basic sciences are being adequately taught, "little thought is being given to the teaching of their application" to the problems of daily practice. As a result, the dental graduate "forgets his early training" in the basic sciences and in practice becomes little more than "a glorified mechanic."

In support of the author's contention that too great emphasis is being placed on reparative procedures by modern dental education, the following comparative report of the Curriculum Survey Committee is added as evidence of the increasing emphasis being given to the mechanics of dentistry as compared to the scientific and preventive:

Average Percentage Distribution of Subjects in the Dental Curriculum in 1941-42 as Compared with the Recommendations of The Curriculum Survey Committee in 1934.

Subjects:	*Recommended in 1934*	*In Operation in 1941-42*
Anatomy, including oral anatomy histology, and embryology	13.05	12.94
Physiology, general and oral	4.39	3.98
Biochemistry	2.93	3.08
Bacteriology	2.19	2.91
Pharmacology and materia medica	1.83	2.14
Pathology, general and oral	4.39	4.22
Medicine, general and oral	3.39	1.65
Dental materials	2.29	2.16
Diagnosis and treatment-planning	2.74	1.94
Radiography	1.42	1.41
Orthodontics	2.56	2.88
Dentistry for children	0.00*	2.33
Operative dentistry	23.63	20.58
Periodontia	0.00	2.10
Pulp-canal therapy	0.46	0.91
Prosthodontia	23.33	25.94
Oral surgery and anaesthesia	2.47	4.96
Public health and hygiene	1.83	1.00
History, ethics, jurisprudence, practice management, and technical composition	3.16	2.87
Physics	1.47	0.00
Organic Chemistry	2.56	0.00
	100.00	100.00

*Included in operative dentistry.

The following are some of the author's conclusions drawn from his review of present-day dental education:

First: "The planning of dental education for the future must include a more important place in the curriculum for basic sciences and their application to preventive practice through a clinical orientation course."

Second: "Subjects such as Oral Diagnosis, Pedodontia, Periodontia and Orthodontia must occupy a more prominent place in the curriculum."

Third: "In order to bring about a state of good dental health for our children it will be necessary for the dental profession to do considerable planning for the future, realizing as it is done, that the dental education of today determines the type of dentist of tomorrow, which in turn governs the dental health of the future." A. H. M.

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IN MEMORIAM

Doctor Rowe Smith

Texarkana, Tex.

October 10, 1952

Dr. Smith was an Associate member of the Texas State Dental Society, having maintained his affiliation with American Dental Association through the Arkansas State Dental Association.

Following the precept that a professional man must be a continuous student, Dr. Rowe Smith set an academic pattern following his graduation from Vanderbilt University Dental School (1923). He completed post-graduate training at Stillman-McCall Clinic, New York City, in 1926, and at Northwestern University (1927); postgraduate certificate in Oral Pathology and Periodontics, University of Vienna, Austria. He was an annual participant in courses of the Colorado Foundation of Dental Science and received a five-year certificate from the California Dental Medicine Seminar.

His presentations before the American Dental Association, the Chicago Mid-Winter Clinic, the Mid-Continent Dental Congress (St. Louis), Canadian Dental Association, Toronto, and state meetings of the Arkansas, Texas, Oklahoma, Nebraska, Louisiana, Tennessee, Mississippi, and South Carolina Dental Associations were well received; and his appearances on programs for city, district and regional meetings were numerous.

His contributions in the field of periodontics are well known, as evidenced by the great demand for his presence as a lecturer and clinician. Those who knew him intimately respected not only his scientific leadership but were captured by his devotion to his fellow man.

While these accomplishments reflect his tremendous stamina and professional excellence, the salient feature of this man was his ability to impart to his patients and his associates the warmth and enthusiasm that could only come from a Christian heart.

The death of Dr. Rowe Smith poses a challenge to those who follow. The challenge is scientific leadership combined with a devotion and understanding of our fellow man.

As long as we accept and meet this challenge, as did Rowe Smith, our profession is secure.

William R. Patterson, D.D.S.
Texarkana, Ark.-Texas.

Reprinted from the Texas Dental Journal, November 1952, page 426.

IN MEMORIAM

Doctor William Oliver Talbot
Fort Worth, Tex.
Feb. 2, 1873 Oct. 14, 1952

Doctor Talbot was one of the Lamplighters for dentistry in the Southwest. He was a strong adherent for dental research, dental education, professional character, and tolerance. Dr. Talbot believed that life was to be used, not placed upon a shelf for the sake of preservation. To this end he lived life fully. He endeavored to make his all count to the uttermost for his beloved profession, and for the happiness of others.

Dr. Talbot was meticulous in matters of detail. When an assignment was given to him, for study and report, there was no question regarding the outcome. Dr. Talbot generously gave of his time and talents in the preparations of reports and papers for dentistry. Material sent to the Journal by Dr. Talbot could be forwarded on to the printer without editing or reviewing.

Dr. Talbot was the possessor of clear vision and ability for practical evaluation, translating mental capacity and vigor into fixed purpose. His contributions to the profession were the true end results of sincerity and faith. He bore the marks and the scars of a progressive leader, long since having learned that one cannot lessen extent of knowledge by giving it away. His life was mellowed by a strong sense of humor, and his kindly smile was always at hand as encouragement to greater friendship.

Dr. Talbot could at all times be depended upon for honest and frank opinions, regardless of circumstances. He had a high sense of respect for the rights of his friends to differ with him.

Our remembrance of Dr. Talbot will never dim in regard to his worthy example of uncompromising honesty; his loyalty to his friends, his profession; and his resolute effort to attain perfection in his daily tasks. The loss of Dr. Talbot is shared by all who knew him.

Doctor Talbot was born at Harperville, Miss. Graduate of Northwestern University Dental School, Dr. Talbot practiced dentistry at Biloxi, Miss., for 15 years; Fort Worth, Texas since 1910. Dr. Talbot was a member of the Miss. Dental Association; the Southern Dental Assoc.; American Society of Orthodontists; Fort Worth District Dental Society; Texas State Dental Society; American Dental Association; American College of Dentists; American Academy of Periodontology.

Dr. Talbot served as secretary and also as president of the Mississippi Dental Assn.; secretary and later as president of the Texas State Dental Society; a member of the Board of Trustees, American Dental Association for 6 years; a member of the Texas State Board of Dental Examiners for two years. Dr. Talbot was a member and elder of the First Presbyterian Church, Fort Worth. He was a member of the York Rite Bodies of Masonry and the Shrine. Dr. Talbot married Miss Rose Dale Andrews, October 4, 1901, in Biloxi, Miss. Three children—Oliver, Green and Mary, together with Mrs. Talbot, survive him.

Reprinted from the Texas Dental Journal, November 1952, page 405-6.

Dr. I. Hirschfeld suggests . . . . .

For many years, periodontists and possibly other practitioners have been plagued with the time many patients take at each sitting to re-fill the drinking cup at the cuspidor. This is particularly so when the operator is cramped for time. Some not only rinse frequently, but use up the full cup at each rinsing. Often five or more minutes are lost at each visit for just re-filling the cup. Multiply this by the number of hours spent at the chair daily and one realizes that he thus loses two to two and a half hours weekly.

Attempts to curb such procedure is not always diplomatic nor successful.

The following very simple method has proven very satisfactory, both to the operator and the patient:

Before placing the paper cup into the holder, the assistant punches a hole (with a regular paper punch purchasable in the stationery store) on the side of the cup, about a half inch from the border or lip. The cup is placed on the

cuspidor platform with the hole *away* from the patient, so as to avoid spilling while rinsing. The faucet is turned on so as to let the water drip into the cup at any desired speed. The purpose of the hole is to prevent the water from reaching the rim and so to simplify handling of the cup.

In this manner, the cup is always sufficiently full for rinsing when it is required.

The advantages from so saving approximately two hours' time a week are fairly numerous, but the operator may best decide on how to utilize it.

Announcements

AMERICAN ACADEMY OF PERIODONTOLOGY

1953 ANNUAL MEETING—SEPTEMBER 23, 24, 25

COMMITTEES

Committee on Scientific Sessions
Robert G. Kesel, *Chairman*
Douglas A. Kerr
B. O. A. Thomas

Program Committee
Frank E. Beube, *Chairman*
Lester W. Burket
John F. Prichard
Dorothea F. Radusch
Harold G. Ray

Properties Committee
Albert Kniesner, *Chairman*
Richard Engel
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Louis Madorsky

Local Arrangements Committee
Marion L. Drake } *Co-chairmen*
Leon M. Wiesstien }
G. P. Bannister
Mildred W. Dickerson*
J. S. McKenzie

Publicity Committee
Howard A. Hartman, *Chairman*
Francis J. Fabrizio
Arthur C. Heibert

Clinic Committee
Evert A. Archer, *Chairman*
S. Parker Gay
Tolar N. Hamblin
Wallace C. Mayo
S. Leonard Rosenthal
Rexford H. Stearns

Entertainment Committee
E. Ball, Jr., *Chairman*
E. Ball, Sr.
Louis Biales
Howard A. Hartman

Registration Committee
Louis Biales, *Chairman*
W. M. Gardner
J. Richard Loughry

Ladies' Activities
Mrs. Wiesstien, *Chairman*
Mrs. Biales
Mrs. Drake
Mrs. Elam*
Mrs. Hartman
Mrs. Heibert
Mrs. Loughry
Mrs. Riffle

*Taking charge of President's Saturday 4 o'clock tea.

ACADEMY OF PERIODONTOLOGY SPRING SEMINAR—APRIL 27-29, 1953 ANN ARBOR, MICHIGAN

1953 PROGRAM IN CHARGE OF DR. EDGAR JAMES

Monday, April 27

8:30- 9:30 Registration in charge of Dr. William Mann

10:00-12:00 Dynamics of Inflammation—Dr. Valey Menkin, Temple University School of Medicine, Philadelphia 40, Pa.

2:00- 4:30 Dynamics of Inflammation

Tuesday, April 28

9:00-12:00 Role of Cells in Inflammation—Dr. W. E. Ehrich, University of Pennsylvania Graduate School of Medicine, Philadelphia 4, Pa.

2:00- 4:30 Pathology of Inflammatory Processes in Gingival Disease—Dr. Hamilton B. G. Robinson, Columbus, Ohio

Wednesday, April 29

9:00-12:00 Classification, Differential Diagnosis and Treatment of Gingival Disease

2:00- 4:00 Continuation of A.M. Discussion—Drs. D. A. Kerr and Sigurd Ramfjord, School of Dentistry, University of Michigan, Ann Arbor, Michigan

Write Dr. William Mann, University of Michigan, Ann Arbor Michigan for details of registration and arrangements.

A.A.O.P. MEETS FEB. 8, 1953

The American Academy of Oral Pathology will hold its seventh annual meeting at the Conrad Hilton Hotel in Chicago on February 8, 1953, immediately preceding the Midwinter Meeting of the Chicago Dental Society.

The program committee, under the able chairmanship of Dr. Hamilton B. G. Robinson, has arranged a most interesting program which is tentatively outlined.

Tentative Program
9:00 A.M.

Dr. Marvin Burstone, University of Illinois
"Histochemical Study of the Irradiated Nuclei"

Dr. Charles Waldron, Washington University
"Giant Cell Tumors of the Jaw"

Dr. Victor Halperin, Ohio State University
"The Occurrences of Fordyce Spots, Fissured Tongue in Median Rhomboid Glossitis in 2,400 Patients"

Dr. Steve Kolas, Ohio State University
"The Occurrence of Torus Palatinus and Torus Mandibularis in 2,400 Patients"

Dr. Hans Schlumberger, Ohio State University College of Medicine
"Comparative Pathology of Oral Neoplasms"

Dr. Myron Aisenberg, Baltimore College of Dental Surgery
"The Histopathology of Ameloblastoma"

Dr. William Shafer, Indiana University
"Ameloblastoma: A Case Report"

Panel Discussion with audience participation and Questions and Answers: Participants—
Dr. Harry Archer, University of Pittsburgh
Dr. Balint Orban, Colorado Springs
Dr. Joseph L. Bernier, Armed Forces Institute of Pathology
Dr. Hamilton B. G. Robinson, Ohio State University

FOURTH MID-ATLANTIC STATES CONFERENCE IN ORAL MEDICINE
SKYTOP CLUB, SKYTOP, PENNSYLVANIA

APRIL 26-MAY 1, 1953

Sunday, April 26, 1953

3:00- 6:00 p.m.	Registration	
6:00- 7:00 p.m.	"Get-acquainted" party	
7:00- 8:00 p.m.	Dinner	

Monday, April 27, 1953

9:00-10:00 a.m.	Introduction of essayists Overview of Seminar	
10:00-11:00 a.m.	Structure of tooth supporting tissues, etiology of periodontal disease	Dr. Paul E. Boyle
11:00-12:00 m.	Systemic etiologic factors in periodontal disease—experimental studies	Dr. H. M. Goldman
7:30- 8:30 p.m.	Pathology of periodontal disease—gingivitis and periodontitis	Dr. Paul E. Boyle
8:30- 9:30 p.m.	Continuous eruption of teeth and area of tooth attachment and infection as factors in periodontal disease	Dr. C. H. M. Williams
9:30-10:30 p.m.	General discussion period	

Tuesday, April 28, 1953

9:00-10:00 a.m.	Electromyographic technic and its use in determining centric relation, abnormal muscular habits, etc.	Dr. R. E. Moyers
10:00-11:00 a.m.	Pathology of periodontosis and the traumatic periodontal lesion	Dr. Paul E. Boyle
11:00-12:00 m.	Objectives of periodontal therapy	Dr. G. R. Lundquist
7:30- 8:30 p.m.	Clinical studies—systemic etiologic factors in periodontal disease	Dr. H. M. Goldman
8:30- 9:30 p.m.	Curettage and packing in periodontal treatment	Dr. C. H. M. Williams
9:30-10:30 p.m.	General discussion period	

Wednesday, April 29, 1953

9:00-10:00 a.m.	Restorative and prosthetic dentistry in the treatment of periodontal disease	Dr. Lewis Fox
10:00-11:00 a.m.	Physical health as it influences periodontal therapy	Dr. H. M. Goldman
11:00-12:00 m.	Surgery in periodontal treatment	Dr. G. R. Lundquist
7:30- 8:30 p.m.	Occlusal equilibration as an aid in periodontal treatment	Dr. C. H. M. Williams
8:30- 9:30 p.m.	Electromyographic studies in occlusal equilibration	Dr. R. E. Moyers
9:30-10:30 p.m.	General discussion period	

Thursday, April 30, 1953

9:00-10:00 a.m.	Restorative and prosthetic dentistry as an aid to periodontal treatment	Dr. Lewis Fox
10:00-11:00 a.m.	Orthodontics as an aid to periodontal treatment	Dr. R. E. Moyers
11:00-12:00 m.	Rationale for pocket elimination	Dr. H. M. Goldman
2:00- 3:00 p.m.	Restorative and prosthetic dentistry as an aid in periodontal treatment	Dr. Lewis Fox
3:00- 4:30 p.m.	Panel discussion by essayists	
6:00- 7:00 p.m.	Cocktail party	
7:00- 8:00 p.m.	Banquet and entertainment	

Friday, May 1, 1953

9:00-12:00 m.	Recreation
12:30 p.m.	Closing luncheon

The fee for this course is $250.00. For further information write University of Pennsylvania School of Dentistry, Postgraduate Division, Philadelphia, Pa.

THE FOURTH ANNUAL BERKSHIRE CONFERENCE IN PERIODONTOLOGY AND ORAL PATHOLOGY

Tufts College Dental School Postgraduate Division will conduct an advanced conference in Periodontology and Oral Pathology under the direction of Dr. Irving Glickman and staff, at Eastover, a resort hotel in Lenox, Massachusetts, June 14 to 18, 1953. The conference will consist of four days of lectures, seminars, and panel discussions regarding clinical problems by outstanding teachers and clinicians in dentistry, medicine, and their related fields.

The curriculum is arranged so that the mornings and evenings are devoted to professional activities, with afternoons open for recreation.

Eastover, a summer resort, is located high in the west ridge of the Housatonic River,

eleven hundred feet above sea level, dominating an unbroken panorama of the Berkshire Mountains. It offers the ideal environment for both concentrated study and recreation.

THE FACULTY OF THE CONFERENCE

COL. JOSEPH BERNIER, D.D.S., M.S.—Chief of the Dental and Oral Pathology Section of the Armed Forces Institute of Pathology, Pathologist to the Registry of Dental and Oral Pathology

LOUIS ALEXANDER COHN, D.D.S.—Assistant Clinical Professor of Dentistry, School of Dental and Oral Surgery, Faculty of Medicine, Columbia University, New York, Associate Professor of Post-Graduate Prosthetics, Tufts College Dental School

IRVING GLICKMAN, B.S., D.M.D.—Professor of Oral Pathology and Periodontology, Tufts College Dental School

ROGER GUILLEMIN, M.D., Ph.D.—Assistant Professor, Institute of Experimental Medicine and Surgery, University of Montreal

LENT C. JOHNSON, M.D.—Chief, Department of Orthopedic Pathology, Armed Forces Institute of Pathology, Washington, D. C.

DONALD KERR, D.D.S.—Professor of Oral Pathology, University of Michigan School of Dentistry

PAUL LOSCH, D.D.S.—Associate Professor of Pediatric Dentistry, Harvard School of Dental Medicine

HARRY SICHER, M.D.—Professor of Anatomy and Histology, Loyola University Dental School, Chicago, Illinois

I. SNAPPER, M.D.—Director of Post-Graduate Education, Cook County Hospital, Chicago, Illinois. Clinical Professor of Medicine, Columbia University Medical School

DAVID WEISBERGER, B.S., D.M.D., M.D.—Associate Professor of Dental Medicine, Harvard School of Dental Medicine

THE CURRICULUM FOR THE CONFERENCE

Diseases of the Tongue, their Diagnosis and Treatment
Bone in Health and Disease
Clinical Implications of the General Adaptation Syndrome
The Diagnosis and Treatment of Gingival Disease
Stomatology in Children
The Effect of Drugs, Tobacco, Alcohol, and Common Food Substances on the Oral Tissues
Principles of Occlusion Pertinent to the Periodontal Problem
Temporo-mandibular Joint Disturbances, Their Etiology, Diagnosis, and Treatment
Clinical Approach to the Correction of Occlusal Disturbances in the Management of Periodontal Disease

For further information write:

BERKSHIRE CONFERENCE
Tufts College Dental School
136 Harrison Avenue, Boston 11, Massachusetts

SEVENTH ANNUAL SEMINAR OF COLORADO DENTAL FOUNDATION JUNE 21-25, 1953

The Seventh Annual Seminar of the Colorado Dental Foundation will be devoted to the Histopathology of the Oral Cavity and will be held June 21-25, 1953, in Colorado Springs.

The 1953 Seminar is being arranged far ahead of the usual time because of the extensive technical preparations that are necessary for this type of program. Each seminarian who has enrolled by March 15th will receive a set of microscopic slides covering the phases of Oral Pathology that are to be discussed during the meeting.

Slide sets will include:

1. Normal Oral Mucosa
2. Disturbances in Keratinization of Oral Mucosa: Hyperkeratosis, Leukoplakia, Intra Epithelial Cancer (different stages and forms), Epidermoid Carcinoma.
3. Normal Bone: Haversian Systems, Lamellated Bone, Compact Bone, Cancellous Bone, Osteoblasts, Osteoclasts.
4. Pathology of Bone: Osteoporosis, Osteosclerosis, Disturbances in Calcification, Disturbances in Formation and Resorption of Bone.
5. Tumors of the Oral Cavity: Carcinoma, Sarcoma, Odontoma, Ameloblastoma.
6. Pulpitis.
7. Periapical Pathology.

A microprojector will be available for projection of the original slides.

Dr. Harry Sicher will discuss: "The World of Histology", "The Lost Dimension", "Correlation of Histology and Gross Anatomy". He will also discuss the Normal Oral Mucosa, Normal Bone and Its Physiology.

Dr. Joseph P. Weinmann will present Pathology of Bone and Periapical Pathology.

Colonel Joseph E. Bernier will deal with disturbances of keratinization and will discuss the clinical importance of leukoplakia. He also will present an up-to-date classification of tumors of the oral cavity.

Dr. Balint Orban will discuss inflammation and its significance, especially in the dental pulp and will present a clinical pathologic outline of oral lesions for differential diagnosis.

POST-GRADUATE COURSES IN PERIODONTICS

Dr. Balint Orban
629 N. Nevada Avenue
Colorado Springs, Colorado

The following courses are offered in June of 1953

Histopathology of the Oral Cavity June 15-19
(Dr. Joseph P. Weinmann, University of Illinois School of Dentistry, lecturing)

Occlusion in Periodontics June 26-30

Two other courses in Periodontics—Theory and Practice and Histopathology of the Supporting Tissues will be offered in the Fall, the dates to be announced.

For information, please write to Dr. Orban at the above address.

BETH ISRAEL HOSPITAL DENTAL DEPARTMENT

The Beth Israel Hospital Dental Department will offer a course in periodontal practice consisting of lectures, demonstrations, and clinics starting March 2 through March

13, 1953. Instruction will be carried out by Drs. Henry M. Goldman, Bernard Chaikin, Lewis Fox, and associates.

Application may be made to the Director of the Dental Department, Beth Israel Hospital, 330 Brookline Avenue, Boston, Mass.

COLLEGE OF DENTISTRY
THE OHIO STATE UNIVERSITY

The College of Dentistry, The Ohio State University, will present the following postgraduate courses during the winter of 1953.

Jan. 19-23 Periodontics—Dr. John R. Wilson
Jan. 26-30 Children's Dentistry—Dr. Lyle S. Pettit
Feb. 16-20 Partial Denture Prosthesis—Dr. Victor L. Steffel
Mar. 2- 6 Endodontics—Dr. J. Henry Kaiser

Each course is given for five consecutive days, Monday through Friday, and the enrollment is limited to approximately ten per class. The fee is $50 per course with the exception of Oral Surgery, Endodontics, and Airbrasive Tehnic for which a fee of $100 is charged.

Further information and application may be secured from the Postgraduate Division, College of Dentistry, Ohio State University, Columbus 10, Ohio.

POSTGRADUATE TRAINING IN PERIODONTIA AND ORAL MEDICINE
NEW YORK UNIVERSITY COLLEGE OF DENTISTRY

The Periodontia Department of New York University College of Dentistry which has been giving postgraduate courses in this subject since 1926, announces its full time course in Periodontia and Oral Medicine for one academic year, September 21st, 1953 to May 21, 1954, leading to a certificate. Half-time two-year courses are also given. Three-weeks refresher courses and ten session (one evening weekly) introductory courses are given beginning Feb. 25, 1953, June 1, 1953 and Oct. 11, 1954. All courses are under the direct supervision of Dr. Samuel Charles Miller.

For information concerning the above or courses in other phases of dentistry write to Secretary, Postgraduate Division, New York University College of Dentistry, 209 East 23rd Street, New York 10, N. Y.

ATTENTION UNDERGRADUATE TEACHERS OF PERIODONTOLOGY

Again in 1953 the American Academy of Periodontology is sponsoring an essay contest and will award prizes of $50, $25 and $10 for the best essay submitted to the editor by May 15, 1953. One year subscriptions will also be given to the students who write the best paper selected by the chairman of the department of periodontology in each dental school. The names of the winners in the local contests should be submitted to the editor of the Journal of Periodontology prior to June 1, 1953.